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PARENTERAL NUTRITION IN THE SURGICAL PATIENT AS PROVIDED FROM GLUCOSE, AMINO ACIDS AND ALCOHOL

THE ROLE PLAYED BY ALCOHOL*

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NUTRITIONAL NEED in the surgical patient is receiving a gradually increasing recognition. The difficulty of providing nutrition to the patient in the immediate postoperative period is incident either to his inability to consume food or to the inability of his gastro-intestinal tract to retain it. However, the surgeon has been able to reduce dietary insufficiency with parenterally administered glucose and amino acids except for the deficiency of calories.

It has become entirely too common to find that the usual postoperative diet consists of no nutrition on the day of operation (1000 cc. 5 per cent glucose—200 calories—is an occasional exception). On the first postoperative day a clear liquid diet is provided (no calories). On succeeding days the routine procedure is to provide general liquids, soft diet, light diet and a full diet. Presuming that the patient eats all that is offered, only the full diet provides adequate calories and protein. The others represent essentially the nutrition received by a starving person.

Table I illustrates the caloric and protein value of a routine hospital diet.

TABLE I

Diet	Composition	Calories	Protein Gms.
Clear diet	Tea, coffee, broth.	0	0
Full liquid diet	Fruit juice, gruel, cereal, cream, sugar, milk, tea, coffee.	903	27
Soft or light diet	Fruit juice, cereal, toast, milk, potatoes, pureed vegetable, pudding, cottage cheese, jello, cooked fruit, scraped beef, ice cream.	1510	41
Full diet	Fruit juice, cereal, cream, sugar, toast, butter, jelly, soup, meat, potatoes, gravy, vegetable, dessert, rice, noodles, eggs, salad, bread, milk.	2259	70

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The normal dietary protein requirement for a healthy individual is accepted by Best & Taylor[2] as approximately one gram per kilogram body weight. This quantity of nitrogen lies about midway between the two extremes as expressed by Chittenden[7] on the one hand and Voit[42] on the other. In terms of nitrogen this would represent .16 Gm. per kilogram body weight. For the sick individual this requirement may need to be doubled. Certainly it cannot be advantageously decreased.

It is therefore evident that a routine hospital diet will not provide the necessary calories and protein for the optimum nutrition of the sick individual. Even if these were provided, many patients would be unable to consume them.

We have made observations on the dietary intake of a number of uncomplicated surgical cases in an effort to learn what nutrition such a patient would voluntarily consume during his hospital stay. The caloric and protein values of the offered and rejected food were determined in each instance. Table II represents a typical example. A caloric and protein deficit is evident.

TABLE II

Mr. G. D. Age 69, height 5'8", weight 185½ lbs. Rt. ing. hernioplasty

	Temp.	Calories Consumed	Protein Gm.	Nitrogen Balance	Weight	Comments
Day of surgery	98.0	0	0	− 8.3 Gm.	185½	Metabolic requirements calculated
1st postop. day	99.8	796	52	− 2.5 Gm.	.....	(Basal + 38%)= 2220 calories
2nd postop. day	99.6	922	33	−20.3 Gm.	.....	
3rd postop. day	98.4	606	30	−22.9 Gm.	.....	
4th postop. day	99.0	1657	45	− 1.0 Gm.	174½	Patient up
5th postop. day	99.0	1034	50	− 5.6 Gm.	176¼	Weight loss 9¼ lbs.

Another patient, in seven postoperative days, ate food containing only 3827 calories, in which there were 114 Gm. of protein, which is scarcely more than one day's requirement.

Numerous investigators[12, 14, 27] have indicated that the postoperative surgical patient is almost always in a state of negative caloric and nitrogen balance. The weight loss which is evident at the end of the hospital convalescence may be a clinical indication of the degree of these negative balances. Daily weighing is suggested when this is possible.

THE VALUE OF NUTRITION IN THE SURGICAL PATIENT

Clinical experience has taught us that the gastro-intestinal tract may not tolerate food during illness or for several days after an operation. We have been prone to accept this as an index of the lack of need for nutrition.

The mere fact that the patient experiences no desire for food does not prove that he does not need it; neither does the ability of a patient to tolerate a period of starvation for several days indicate any physiologic reason why he should do it or that it is beneficial to him. On the other hand, numerous

studies suggest that a lack of adequate nutrition begins to make itself evident in impaired tissue synthesis within a few hours.[3, 4, 6, 43]

PROTEIN

Protein is the only source from which nitrogen can be obtained. All body tissues require nitrogen for their growth. If nitrogen is not provided from food, it will be taken from other protein-containing body tissues to be utilized in the healing or repair of tissues which are in more urgent need of it. Clark,[6] as early as 1919, indicated the importance of protein for rapid wound healing. Ravdin[16, 33-35] demonstrated that without adequate amounts of essential amino acids cellular repair and growth cannot take place. Madden, Bassett and Remington[22] pointed out that a chronically unhealed wound in a patient depleted of protein healed rapidly after the patient was placed on a high protein diet.

CALORIES

A definite number of calories are required by every individual in order to maintain life. These calories represent the basal calories. They can be calculated fairly accurately on the basis of the height, weight, sex and age of the individual or they can be accurately measured with the aid of a Jones metabolic apparatus. Table III shows the correlation between the calculated and determined metabolic requirements in ten different patients. From these figures it is evident that the calculated metabolic requirements and the measured metabolic requirements are close enough for clinical use.

TABLE III

Patient	E.J.	A.N.	J.H.	E.H.	P.H.	D.A.	N.R.	H.W.	W.C.	G.R.
Calculated basal caloric requirements*	1449	1300	1572	1701	1390	1465	1728	1589	1853	1488
Fever requirements (8% per degree)	81	....	....	272	....	....	552	....	444	153
Specific dynamic action requirements 8%	135	104	125	136	111	117	138	127	148	119
Bed activity 30%	506	390	472	510	417	439	518	476	556	446
Calculated total requirements	2171	1794	2169	2619	1918	2021	2936	2192	3001	2801
		1711†								
Measured metabolic requirements (Jones)	2422	1686	2527	2653	1853	1853	2603	1986	2925	2780

* Calculated on the basis of the height, weight, age and sex of the individual and with the aid of the deBois charts.

† Repeated.

If basal calories are not provided from food, they are obtained from body tissues as manifested by a loss in body weight.

Cannon[4] has shown by animal experiment that the process of tissue synthesis requires the simultaneous availability of all essential amino acids as well as energy (calories). In his experiments on rats he showed that a deficiency in either calories or protein could cause a delay in the recovery of protein-depleted rats; that the tissue synthesis proceeded in direct relation to the amount of calories, or in direct relation to the amount of protein when the other element of food was provided in adequate amounts. When both elements

of nutrition (protein or calories) were provided in adequate amounts, healing again occurred rapidly.

GLUCOSE

The most readily available intrinsic source of calories is body fat, but in order to prevent the development of acidosis, glucose must also be available. In starvation, the reserve store of glycogen in the liver is rapidly utilized and thereafter the necessary glycogen is resynthesized from amino acids.

Glucose must be available in order that amino acids will not be converted into glycogen and thereby lose their nitrogen value for tissue repair. In addition to contributing to the combustion of fat, glucose also has value in providing a direct source of energy.

FATS

Though in this study the calories from fat have been substituted by alcohol, it is not desired to indicate that fats are dispensable. For the moment, until more adequately utilizable fats are available for parenteral use, the fats are more dispensable for short periods of time than the other metabolic elements.

Fats provide transportation for fat soluble vitamins. Fats also provide the basic elements for certain unsaturated fatty acids which cannot be resynthesized in the body. The prolonged absence of these fatty acids will produce a delay in normal growth and may cause abnormal skin conditions. In the short periods of postoperative convalescence these factors play no significant role in the healing process.

The principal difficulty of providing adequate nutrition parenterally has been that of providing adequate calories.[1, 7, 18, 33-35]

Fats provide large quantities of calories. These calories can be utilized as energy and can spare the use of protein and carbohydrates for that purpose. Madden, *et al.*[22] state that parenteral feedings are not limited by nitrogen intake, but by the lack of suitable caloric intake. They state that fat emulsions have, so far, failed to be suitable sources of calories.

Ravdin[19, 34, 35] also tells us that calories protect protein. Altschuler[1] advises that enough calories must be provided lest amino acids be used for caloric energy instead of for tissue repair.

VITAMINS

Vitamin B complex and Vitamin C are also needed for tissue synthesis and repair.[17, 24, 41] It is evident that all of the essential metabolic elements should be provided simultaneously and in adequate amounts in order that the optimum benefit can be obtained from each, and that the best nutrition for wound healing can be provided.

THE NUTRITIONAL VALUE OF ALCOHOL

It is well recognized that alcohol has caloric value. Whether or not these calories can be utilized for work energy has been debated. It is very possible,

however, that alcohol calories can be utilized as energy for normal metabolism when the excessive demands of work energy are not required.

The experimental work done on the energy value of alcohol has been done without adequate consideration for the carbohydrate and protein requirements. If these nutritional elements are also taken into consideration, it is very much more likely that alcohol calories can be substituted for fat calories without significant loss to the individual. Investigative work disclosing the bad effects of alcohol and other hepatotoxic substances have demonstrated that these undesirable effects cannot be reproduced when adequate nutrition is provided.[9-11, 20, 21, 32, 40, 44]

Higgins[18] appears to have shown that alcohol can be substituted for carbohydrates and fats as indicated by a study of the respiratory quotient. He found that the respiratory quotient was lowered when alcohol was fed in conjunction with other foods. He further mentions that although alcohol is not readily available as a food, it will be burned in preference to the sugar when both are taken together. This was also demonstrated by Pogel, Grezino, and Durgin[31] in 1913. Moore and Karp[25] concluded that the intravenous use of 5 to 10 per cent alcohol could increase the caloric intake and thereby have special value in cases suffering from inanition. They maintain that alcohol can protect carbohydrate, protein, and body fat because it is readily available energy which cannot be stored. Coffey[8] has also spoken of the caloric value of intravenous alcohol. He recommends its use in order to prevent the utilization of reserve carbohydrate and protein. Mendel and Hilditch[23] stated that alcohol could be of definite service in preventing tissue waste in those patients who were suffering from high fever and undernourishment, who otherwise would call upon their own body tissues to make up for the caloric deficiencies. When there is difficulty in supplying nourishment with ordinary food, they state that alcohol will be of considerable value as a substitute for these.

METHOD OF INVESTIGATION

In this study alcohol has been substituted for fat because of its relatively large caloric value and because it is commercially inexpensive. Our studies have been undertaken on clinical patients admitted to the St. Barnabas Hospital for diagnosis or operation. The studies were discontinued if they interfered with or disturbed in any way the proper care of the patient. Whenever detailed studies were done, the patient was advised of these in order to obtain co-operation and to allay his fears with regard to costs, number of tests, etc.

As far as we have been able to learn from the medical literature, though each of these nutritional elements have been given separately, all three have not been administered simultaneously prior to the time that we first used them two years ago. We have now used a mixture of glucose, amino acids and alcohol* routinely in more than 600 postoperative surgical patients in the past two years. It has been our aim to provide parenterally the full nutritional and

* Commercially prepared amigen, aminosol or travamin, to which the alcohol is added into the commercially prepared bottles through the rubber stopper.

caloric requirements of the individual until he is able to consume food without difficulty and without discomfort.

It has become our routine in the postoperative surgical patient to administer immediately after operation 1000 cc. of fluid containing amino acids 5 per cent, glucose 5 per cent and 60 cc. of 98 per cent alcohol.* This is given slowly over a period of four hours. During this period of time the patient usually sleeps or dozes. Morphine is rarely given and rarely required. In the late afternoon and evening another 1000 cc. is given. Therefore, within the first 12 hours after operation the patient has received 100 Gm. of amino acids, 100 Gm. of glucose and 120 cc. of alcohol; a total of 1472 calories. On the following morning the patient is offered food in accordance with his desires, and if enough is consumed no further parenteral nutrition is given. If the patient does not eat well, parenteral nutrition is supplemented to the point of the calculated metabolic requirements.

We have also used this method of nutrition to prepare poor surgical risk patients for operation.

We contend that it is desirable to satisfy at all times in all surgical patients complete nutritional requirements (calories, proteins, carbohydrates, vitamin B and C, and electrolytes), and to maintain a positive nitrogen balance, body weight, and fluid balance.

BLOOD ALCOHOL LEVEL

Haven Emerson[15] has observed that when a specific quantity of alcohol is ingested by mouth its highest concentration in the blood appears within the first one and one-half or two hours. Thereafter it gradually diminishes, requiring 15 or 16 hours before all traces of alcohol have disappeared from the blood.

Our studies on the administration of alcohol, given intravenously alone, or in conjunction with glucose and amino acids, indicate considerable variation in different individuals. Since we found considerable variation in the blood alcohol concentration among a number of patients, we conceived the idea that the rate of utilization, and therefore the rate of administration, might be related to the caloric demands of the individual. Thereafter we administered alcohol on the basis of its caloric value in relation to the caloric demands of the patient.

The basal caloric requirements were at first determined on the Jones metabolism apparatus; later calculated on the basis of height, weight, age, sex, and with the aid of the duBois charts. Eight per cent of the basal caloric requirements were added for specific dynamic action, 30 per cent for bed activity and 8 per cent for each degree rise of fever, the sum of which represented the caloric requirements of the patient. A comparison of these two methods for determining the metabolic requirements of the patient indicated the latter to be accurate enough for clinical use (See Table III). When the caloric require-

* Commercially prepared amigen, aminosol or proteolysate, to which the alcohol is added into the commercially prepared bottles through the rubber stopper.

ments of the patient were used for determining the rate of administration of alcohol, some fairly definite findings seem to be repeated in most instances.

When the alcohol was given at such a rate that its caloric value was equivalent to approximately two times the caloric demand of the patient, the blood alcohol rose to a fairly constant level within one hour. Thereafter, if the same rate of administration was continued, the blood alcohol level rose less rapidly, demonstrating a tendency to level off (See Fig. 1, first and second hour). When the rate of administration was then decreased so that the alcohol calories were administered at approximately the caloric demands of the individual, the alcohol level of the blood then ceased to rise (See Fig 1, second to sixth hours). In this instance there was a slight depression of the blood alcohol level after the third hour.

A similar blood alcohol level may be observed in Figure 2 in which, after the first hour, the rate of administration was decreased to slightly above the caloric demands.

Figure 3, in which the rate of administration of alcohol calories was essentially the same as the caloric requirements of the individual, illustrates an insignificant elevation in the blood alcohol level. This suggests that the alcohol is metabolized at approximately the rate of the caloric demands.

In one instance (Fig. 4) the alcohol administration was continuously maintained slightly above the caloric requirements, and the blood alcohol level rose slowly up to .07 Gm. per 100 cc. within the first four hours and for the next four hours remained almost constant. From a practical standpoint this rate and constancy of administration is entirely satisfactory.

We have observed that in order to obtain a sedative and analgesic effect from alcohol it is necessary to raise the blood alcohol level to .07 to .1 Gm. per 100 cc. Mild sedation and analgesia are provided and the caloric demands of the average adult individual are maintained. Sixty cc. of alcohol, mixed with aminosol, given uniformly over a period of four hours, provides the optimum clinical rate of administration in most instances. If the amino acid preparations are administered more rapidly, nausea and other undesirable effects may be produced in a considerable percentage of patients.

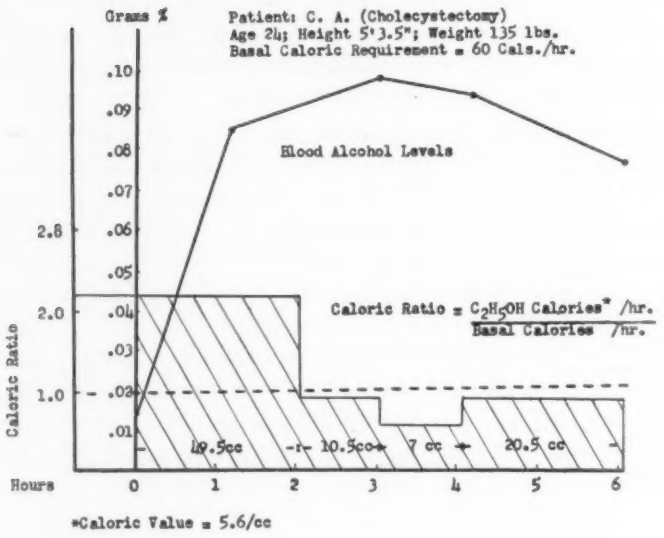
In the composite graph (Fig. 5) dealing with the blood alcohol levels in a number of different individuals, each receiving intravenous alcohol calories at a ratio approximately two times their metabolic requirement, it is seen that the alcohol levels rose rapidly during the first two hours and thereafter had a tendency to level off despite the constant and uniform administration of alcohol.

The blood alcohol level can be raised or lowered if the rate of administration is increased or decreased. However, the rise in the blood alcohol level is not necessarily in proportion to the increased rate of administration.

THE EFFECT OF ALCOHOL ON BASAL METABOLISM

The preceding studies suggest that the alcohol is metabolized somewhat more rapidly when the blood alcohol level reaches a higher figure (.08-.1 Gm.

FIG. 1



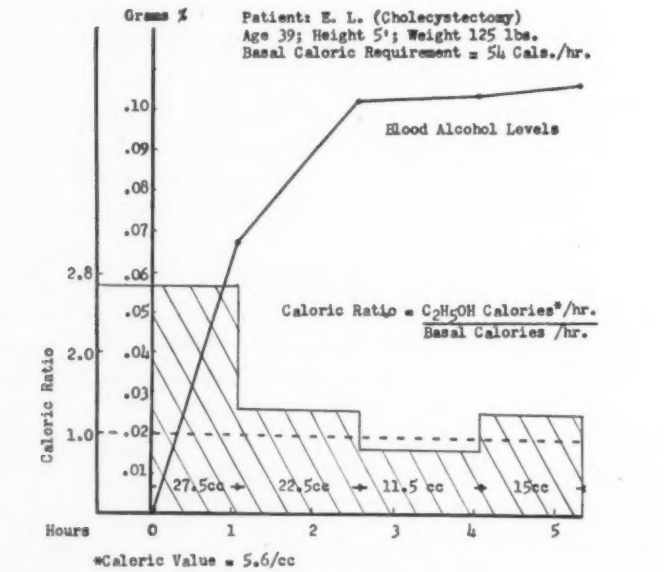


FIG. 2

FIG. 1.—The caloric ratio represents the number of alcohol calories administered per hour in relation to the basal caloric requirement of the patient.

$\frac{\text{Alcohol Calories/hr.}}{\text{Basal Calories/hr.}} = \frac{60}{60} = 1$. This figure is indicated by the dotted line. In this instance the patient received alcohol calories for the first two hours, slightly in excess of two times the caloric requirement. Thereafter the alcohol calories were administered at approximately the caloric de-

(Legend continued on opposite page.)

100 cc.) than when there is less circulating alcohol. If this were not so, we would expect the blood alcohol level to rise steadily in a ratio proportionate to the duration and rate of administration. In view of the fact that the blood alcohol levels ceased to rise at the same rate as was observed during the first hours, we presumed that alcohol given intravenously increases the caloric demands, thereby causing a more rapid metabolism of circulating alcohol so as to account for the smaller rises in the blood alcohol levels as determined from hour to hour.

This was further suggested by conducting a Jones metabolic determination before and during the administration of alcohol. The determinations were made on the time consumed in utilizing one liter of oxygen, calculating from this the basal caloric requirements for 24 hours. The experimental test was done immediately after the control test. In order to produce similar circumstances, intravenous saline was given for the control. The saline solution, connected to the intravenous needle, was substituted by the alcohol solution in each instance and the solution allowed to run for one-half hour before making the experimental test, keeping the patient otherwise under basal conditions. The following result was obtained:

Control metabolic rate	Experimental metabolic rate
I. V. saline being given	I. V. alcohol being given
1112 calories	1420 calories (28 per cent increase)

In like manner, doing similar control tests using glucose, the caloric requirements were as follows:

Control metabolic rate	Experimental metabolic rate
I. V. saline being given	I. V. glucose being given
1324 calories	1389 calories (5 per cent increase)

When amino acids were used, the caloric requirements were considerably increased:

Control metabolic rate	Experimental metabolic rate
I. V. saline being given	I. V. amino acids being given
1049 calories	1589 calories (51 per cent increase)

These metabolic determinations indicate that the glucose and amino acids each caused an increase in the metabolic requirements, essentially in proportion to their specific dynamic action.

It is possible that other factors than the specific dynamic action of alcohol contribute to the greater caloric requirement as the alcohol content of the

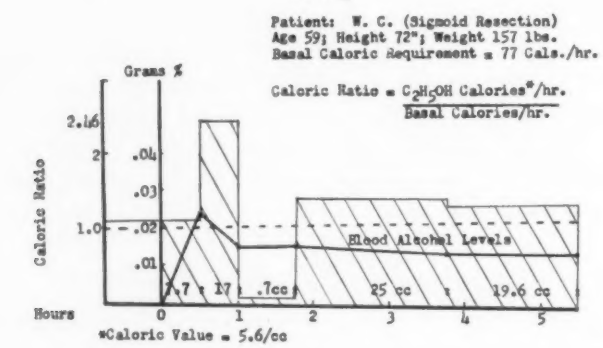
mands of the patient. (See upper graph.) The cubic centimeters indicate the rate of administration of alcohol in volume. Sixty cubic centimeters of 95 per cent alcohol were put in 1000 cc. of normal saline and administered intravenously. The rate of drip was adjusted so as to provide the desired cubic centimeters and caloric ratio of alcohol. The blood alcohol level is recorded in grams per 100 cc.

FIG. 2.—See Figure 1 for explanation of the plottings. In this instance the caloric ratio was 2.8 times the caloric requirement for the first hour. Thereafter it was reduced to slightly above the caloric ratio. The blood alcohol rose rapidly and then leveled off to the end of the period of study.

blood rises. It may cause a reflex stimulation of insulin secretion as suggested by Haven Emerson, and thereby a more rapid combustion; or there may be an increase in the caloric demands incident to an increased capillarity. Some such factors must account for the leveling off of the alcohol curve as illustrated in Figures 1 and 2.

A presumed specific dynamic action of alcohol has been indicated as an objection for using alcohol for its caloric value.[46] From a practical standpoint, such an objection seems no more reasonable than it would be to object to the use of protein because of its still greater specific dynamic action. The specific

FIG. 3



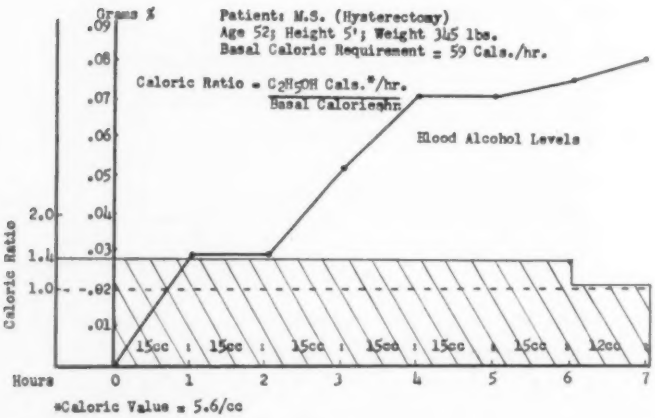


FIG. 4

FIG. 3.—(See Figure 1 for explanation of plottings). This shows the very minimal rise in the blood alcohol level when the rate of administration is kept low. In this instance the rate of administration remained essentially at the caloric demand throughout the period of study except for a short period of time in the last half of the first hour. This rate of uniform administration is used when only the caloric value of alcohol is desired.

FIG. 4.—(See Figure 1 for explanation of plottings). This shows the slow rise in the blood alcohol level when the rate of administration was kept constantly slightly above the caloric requirements. This is the rate of administration most commonly used.

dynamic action of alcohol has been established as approximately 7 per cent.[5] Further substantiation is being pursued.

THE NITROGEN SPARING EFFECT OF ALCOHOL

Our studies relative to the substitution of alcohol calories for glucose calories indicate an increase in the nitrogen output and a decrease in the

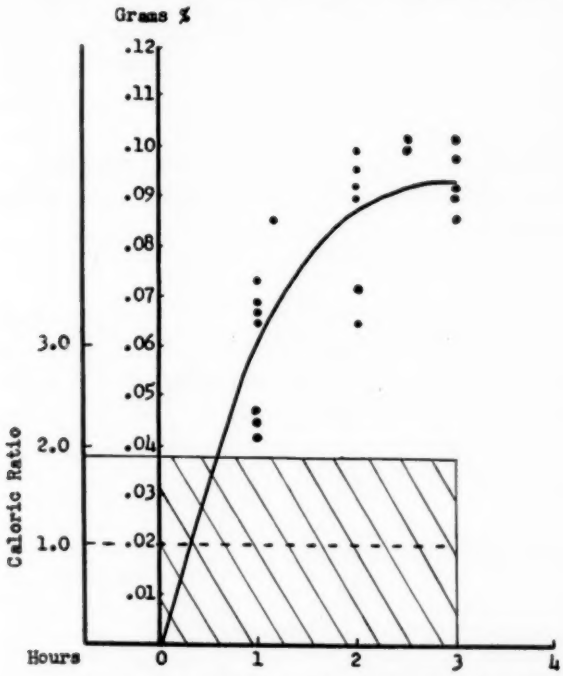


FIG. 5.—Composite graph illustrating the rise in the blood alcohol levels. Alcohol calories were administered uniformly for three hours at the caloric ratio of 1.9 x the basal caloric requirement. This was done in seven different individuals. Blood alcohol levels were determined at intervals of one hour. Each blood alcohol level is indicated by a dot. The black line indicates the rise in the blood alcohol level. This illustrates that the blood alcohol level does not rise at a uniform rate, but has a tendency to level off when the alcohol concentration in the blood reaches .07-.1 Gm. per 100 cc. There may, however, be considerable variation in different individuals as indicated by the scatter of the dots.

nitrogen balance, suggesting that alcohol calories alone cannot completely replace glucose calories. This is indicated by comparing Periods I and II, Table IV, wherein the nitrogen balance becomes negative when alcohol alone is used (Period II). This suggests that glucose is necessary for the economic utilization of amino acids.

On the other hand, when part of the calories is provided by glucose and part by alcohol, an advantage is obtained and a slightly higher nitrogen balance

develops as illustrated in Period III, Table IV. Had the percentage of calories from protein been kept at 37 per cent in Period III as in Periods I and II, it is reasonable to presume that the nitrogen balance would have been still higher despite the negative caloric balance in each of the periods.

TABLE IV

Mr. L. W., age 42, wt. 100, height 5'9"	Period I 4-day Average	Period II 4-day Average	Period III 4-day Average
I. V. amino acid calories	585	608	500
I. V. glucose calories	985	0	500
I. V. alcohol calories	0	1008	840
Total calories given	1570	1616	1840
% calories from protein	37%	37%	27%
Nitrogen output, Gm.	23.2	29.6	16.8
Nitrogen intake, Gm.	23.4	24.3	20
Nitrogen balance, Gm.	+.2	−5.3	+3.2

Metabolic requirements calculated 2100.

In Table V, where the patient was kept in positive caloric balance and the intake of nitrogen remained the same in each period, a more positive nitrogen balance was maintained when glucose, amino acids and alcohol were given (Period I) than when all the calories were derived from glucose and amino acids alone (Period II).

TABLE V

Mr. H., age 53, weight 128 lbs., height 5'8"	Period I 3-day Average	Period II 2-day Average
I. V. amino acid calories	600	600
I. V. glucose calories	1000	1500
I. V. alcohol calories	672	0
Total calories given	2272	2100
% calories from protein	26%	28%
Nitrogen output, Gm.	19.9	22.9
Nitrogen intake, Gm.	24	24
Nitrogen balance, Gm.	+4.1	+1.1

Metabolic requirements calculated 2072 C.

Many other nitrogen balance studies have convinced us that alcohol has a very definite value in providing calories which must otherwise be provided from glucose or amino acids. If these nutritional elements must be used to provide calories, they are thereby eliminated from being used in tissue synthesis.

Our studies further indicate that it is difficult to maintain a strong positive nitrogen balance without providing full metabolic (caloric) requirements unless an over-abundance of nitrogen is provided. This is illustrated in Table VI.

Each period in Table VI represents a series of several days during which time the patient received essentially the same caloric and nutritional elements within the consecutive series of days. The quantity of the various dietary elements was changed in each period. It will be noted that the nitrogen intake

was adequate in all periods in that it was in excess of .16 Gm. per Kg. body weight.

In Periods II and IV the caloric intake was 45 to 50 per cent below the calculated metabolic requirements and in both periods there was a strong negative nitrogen balance despite the adequacy of nitrogen intake.

In Period I the caloric intake was 10 per cent below the metabolic requirement, but the nitrogen intake was high. This resulted in nitrogen equilibrium. In Period III the caloric intake was 15 per cent below the metabolic requirement, but the nitrogen intake was still greater, resulting in a still stronger positive nitrogen balance. Blood transfusions contributed to the nitrogen intake in this period.

The question then arises as to whether or not the intake of nitrogen can be given full credit for the difference in the nitrogen balance. Periods V and VI, another patient receiving identical quantity of nitrogen but different caloric

TABLE VI

Mr. C.	High Nitrogen Cals. 10% Below Requirements	Adequate Nitrogen Cals. 50% Below Requirements	High Nitrogen Cals. 15% Below Requirements	Adequate Nitrogen Cals. 45% Below Requirements	High Nitrogen Mr. L. Wilson	
Age 59, Height 6' Weight 71 kg.	Period I 3-day Average	Period II 7-day Average	Period III 4-day Average	Period IV 4-day Average	Period V 2-day Average	Period VI 3-day Average
Calories:						
I. V. blood	.....	.....	281	.....	.....	.....
I. V. amino acids	600	257	472	400	600	600
I. V. glucose	900	286	785	425	600	1000
I. V. alcohol	1176	392	798	785	1008	.....
Oral	.....	536	.....	.....	.....	.....
Total calories given	2676	1471	2336	1610	2208	1600
Calories required	2960*	2910*	2700*	2878*	2107	2107
Caloric balance	−10%	−50%	−15%	−45%	+5%	−25%
Protein:						
Protein calories	600	328	772	400	600	600
Protein cal. %	22%	22%	32%	24%	27%	37%
Oral protein, Gm.	.....	18	.....	.....	.....	.....
I. V. protein, Gm.	150	64	193	100	150	150
Total protein, Gm.	150	82	193	100	150	150
Total Nitrogen output	24.37	22.9	22.83	26.8	17.8	27.2
Total nitrogen intake.	24.00	12.9	31.14	16.0	24.	24.
Per Kg. body weight	.34	.18	.43	.22	.34	.34
Nitrogen balance	−.37	−10.0	+ 8.31	−10.8	+6.2	− 2.8

* The caloric requirements vary depending upon changes in body temperature.

values, discloses a positive nitrogen balance when the calories are adequate and a negative nitrogen balance when the calories are decreased by eliminating the alcohol.

These studies, as well as other studies,[23, 30, 36, 37] seem to indicate that even though it may be possible to obtain a positive nitrogen balance if enough

nitrogen is given, it becomes more practical to maintain a positive nitrogen balance by supplying some extra calories with the use of alcohol.

THE EFFECT OF ALCOHOL ON THE GASTRIC SECRETION

Most studies of gastric function after alcohol ingestion have been done on the basis of its local stimulating effect on the gastric mucosa. These studies have resulted in the alcohol meal as a method of studying gastric function, and have shown that alcohol stimulates the flow of gastric juice and hydrochloric acid.

Wolf and Wolff[45] observed an increase of gastric blood flow, acid secretion and motor activity in the stomach after introducing whisky (90 proof) into the stomach. The maximum effect took place in 45 minutes and in 90 minutes the values had returned to normal. They were of the opinion that this effect was systemic and not local, because the rise was not observed until the drug had had time to be absorbed.

Newman and Mehrtens[28, 29] observed a rise in the gastric acid when alcohol was given intravenously. Dragstedt, Gray, Lawton and deArellamon[13] were of the opinion that gastric secretion was stimulated after the use of alcohol because of its tendency to liberate histamine. The histamine gastric juice and the alcohol gastric juice are, however, somewhat different in their composition and the effect of alcohol can be suppressed with atropine more than can be done when histamine is used.

Our studies with the intravenous administration of alcohol at a constant rate for four hours indicate that gastric acids are increased during the period of administration and that the flow of gastric acids again ceases when the administration is stopped. The same effect was observed, however, when glucose and amino acids were given intravenously. The significance of this variation in the gastric acidity in patients who are receiving parenteral nutrition and no nutrition by mouth is not evident at the moment. These studies are being pursued further.

MISCELLANEOUS EFFECTS OF INTRAVENOUS ALCOHOL

Many authors have mentioned the value of alcohol as a sedative and analgesic. We have used it with this in mind for the first few hours after surgery and have thereby almost completely eliminated the need for morphine. The side effects of morphine are undesirable and definitely increase the incidence of postoperative sequelae as illustrated by Strickler and Rice.[38]

Competent observers have maintained that alcohol does not produce symptoms of intoxication in patients suffering from high fever. We have observed symptoms of intoxication when the rate of administration was inadvertently increased too rapidly. On the basis of our blood alcohol studies, it would appear that there is a wide range of safety between a therapeutic dose, an intoxicating dose and a lethal dose.

Sterling Mueller[26] mentions that patients with paralytic ileus or peritonitis are benefited from intravenous alcohol. We have observed a definite decrease in the incidence of paralytic ileus and gas pains, but had thought it due more

to the elimination of morphine and the better nutrition of the patient than to any specific effect of alcohol.

E. T. Reiman[39] suggests that the use of intravenous alcohol postoperatively safeguards against the possibility of atelectasis and other pulmonary complications. Moore and Karp[25] report that patients who receive intravenous alcohol experience a feeling of well-being and a loss of anxiety. They have observed no significant vomiting, acidosis or headache from the intravenous use of alcohol. We have observed this feeling of well-being in our patients to the extent that it is the usual experience to have the patient ask to be allowed up within 24 hours after operation. Part of this feeling of well-being, we believe, arises from the fact that the patient has suffered no 24-hour period of starvation, which in itself would be sufficient to delay the convalescence.

COMMENTS

It would be extremely difficult statistically to evaluate a series of adequately nourished patients as against a group whose nutrition is provided only in proportion to their ability to eat. No one would assert that less well-nourished patients could not get well, nor that they cannot be made early ambulant, nor that their wounds would not heal. It is sufficient to say that just because a patient can get well under inadequate nutrition is no reason why he should be starved postoperatively, if nutrition can be provided, any more than it is reasonable for a healthy, hard working man to starve for a few days.

Adequate nutrition is important in the postoperative patient in order that his healing tissues will have available all of the necessary nutritional elements. These nutritional elements consist of glucose, amino acids, calories, vitamins, electrolytes and fluids. To expedite the healing process a strong positive nitrogen balance rather than a mere nitrogen equilibrium is desirable. Certainly a negative nitrogen balance seems undesirable.

Our clinical observations have convinced us that patients who are provided full nutritional requirements feel better, their wounds heal more rapidly, they suffer less discomfort, they are infinitely more easily ambulated and they experience a shorter postoperative convalescence at home than was observed in our patients two years ago when this regimen had not been instituted. We have repeated these studies so many times and in such a large variety of combinations that we feel justified in proclaiming the benefits which we have illustrated in these separate protocols and graphs.

CONCLUSIONS

The routine hospital diet for a postoperative patient does not provide adequate calories or protein. The postoperative patient is unable to consume by mouth an adequate quantity of food and this should, therefore, be supplemented with parenteral nutrition to the point of providing the full caloric and nutritional requirements.

Postoperative patients obtain a feeling of well-being and appear to have a more satisfactory convalescence when they are provided full nutrition beginning immediately after surgery.

When alcohol is given parenterally in a caloric ratio proportionate to approximately two times the caloric demands of the patient, the blood alcohol level rises rapidly during the first hour, after which it has a tendency to level off. Thereafter, if the rate of administration is decreased to approximately one and a half times the caloric demand, the blood alcohol level remains fairly constant at .08 Gm. per 100 cc. This blood alcohol level produces mild sedation and analgesia, reducing thereby the need for morphine.

Approximately 15 cc. of 98 per cent alcohol per hour in parenteral fluids will provide a practical clinical rate of administration for the average adult individual.

A positive nitrogen balance can be attained parenterally in some instances despite a negative caloric balance if an adequate amount of nitrogen is given. A strong positive nitrogen balance can be more consistently obtained, however, when adequate calories are available from extraneous sources.

A positive nitrogen balance can be more easily maintained when glucose and amino acids are supplemented with alcohol than when those two nutritional elements are given alone.

These studies indicate that the readily available calories derived from alcohol have the ability to spare nitrogen and to that extent can provide energy which must otherwise be obtained from body reserves.

Alcohol is able to provide, in parenteral nutrition, the heretofore missing calories.

A solution of glucose, amino acids and alcohol supplemented with vitamins B and C can provide, parenterally, the essential elements of nutrition, *i.e.*, carbohydrate, nitrogen, calories and vitamins. Electrolytes can be added as needed.

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ACUTE APPENDICITIS IN A RURAL COMMUNITY*

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INTRODUCTION

THE GREAT MAJORITY of articles on acute appendicitis have thus far come from university medical centers or large metropolitan hospitals, very few presenting the subject from the viewpoint of the small hospital or rural center. In 1938 a report[1] was made of the patients with acute appendicitis operated upon at the Mary Imogene Bassett Hospital during the years 1930 through 1937. Our purpose in the present paper is not only to bring the report up to date, comparing the past and current series as to changes in therapy, but also to point out the special problems of appendicitis as seen in a rural population in contrast with those encountered in a large urban area.

Environment of the Mary Imogene Bassett Hospital. The hospital is situated in Cooperstown, Otsego County, near the center of New York state, a village of about 3000 inhabitants. Otsego and the surrounding counties are largely rural in character, the majority of the people depending on dairy farming for their livelihood.

The high percentage of people in the older age groups among the population of this area should be especially noted in relation to the patients seen at the hospital. Dr. Joseph V. DePorte,[2] in speaking before the Conference on Rural Medicine held in Cooperstown in 1938, showed that while it was recognized that the proportion of older people was in general increasing, rural Otsego County was unique when compared with the rest of New York state in having 16.5 per cent† of its population already over 60 years of age—a percentage of older people which would not be reached by the rest of the state until 1965. In short, the group of patients seen in the Bassett Hospital today resembles in age that which may be found some 20 or 30 years hence in hospitals of New York State at large. The significance of this fact will be stressed in discussing the age incidence of appendicitis in this hospital and the consequent seriousness of the disease.

The distance of the patients, in many instances, from the hospital, as shown on the map (Fig. 1), might naturally be expected to play an adverse role in the speed with which they receive surgical care. As a matter of fact however, upper New York State is served by an excellent system of roads which are kept open even in the most severe winters, and distance does not prevent prompt hospitalization, as is shown by Figure 5.

* Read in part before the joint meeting of the surgical departments of the Presbyterian Hospital of New York and the Mary Imogene Bassett Hospital in Cooperstown, June, 1948. Submitted for publication May, 1949. Data based on figures from the census of 1930.

† Based on figures from the census of 1930.

CLASSIFICATION OF CASES

All patients with acute appendicitis operated upon at the Bassett Hospital from 1930 through 1948 are included in this report. Table I shows the total number. It will be noted that since the presentation in 1938 of 182 cases, 358 patients with acute appendicitis have been operated upon, giving a total of 540 cases.

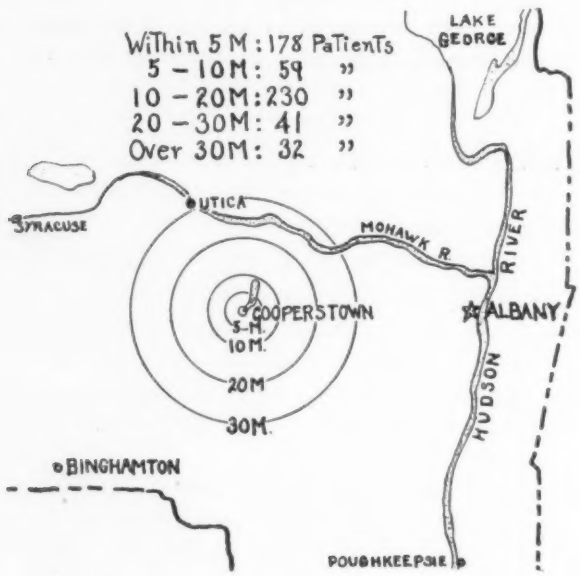

FIG. 1.—Location of Cooperstown in New York State and the approximate distance the patients with acute appendicitis had to travel to reach the M. I. B. Hospital.

TABLE I.—*Acute Appendicitis at the M. I. B. H. 1930–1949.*

	Number of Cases
Series I (1930–1938)	182
Series II (1938–1949)	358
Total	540

The diagnosis of acute appendicitis has in all instances been verified by microscopic examination of the appendix, and where the opinion of pathologist and surgeon have been at variance the judgment of the pathologist has been accepted as final.

The cases have been grouped as nearly as possible after the method of Schullinger,[3] who set up five classes: simple acute appendicitis; acute appendicitis with acute local peritonitis; acute appendicitis with peritoneal abscess; acute appendicitis with acute diffuse peritonitis; and acute appendicitis with

acute progressive fibrinopurulent peritonitis. We have omitted the last class, listing cases which might have fallen into this group as acute appendicitis with acute diffuse peritonitis. Schullinger pointed out that there were three chief difficulties in making a satisfactory classification: first, when the operation was properly done, appendicectomy was a local procedure and the abdomen was not explored; second, there were fine gradations between types which were subject to varying interpretation by different surgeons; and third, the author was forced to make the final classification, basing it (when he was not also the operator) upon what had been recorded on the chart.

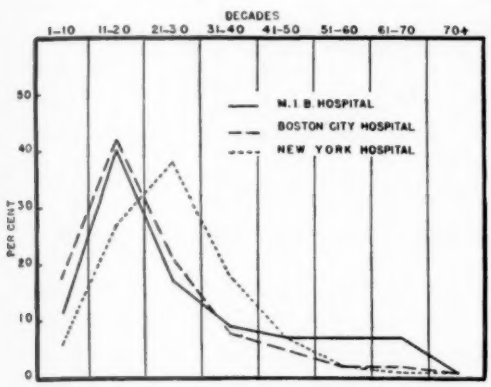

FIG. 2

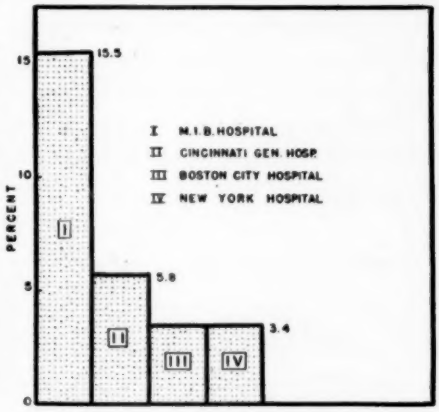

FIG. 3

FIG. 2.—Distribution according to age decades of patients with acute appendicitis at the M. I. B. Hospital, the Boston City Hospital and the New York Hospital. It will be noted that after the fourth decade the Bassett Hospital curve flattens out, while the others continue to drop, and that in the sixth and seventh decades the divergence is striking.

FIG. 3.—The percentage of patients over 50 years of age with acute appendicitis at the M. I. B. Hospital, the Cincinnati General Hospital, the Boston City Hospital and the New York Hospital.

In studying the cases here reported, it was found that simple acute appendicitis and acute appendicitis with peritoneal abscess were usually well recorded in the operative note; and that the most difficult problem was to distinguish between acute local peritonitis and acute diffuse peritonitis. Peritonitis was therefore classified as "acute diffuse" when the following conditions were fulfilled: when tenderness, spasm and rebound pain were present over the entire abdomen; when the clinical picture was consistent with this diagnosis; and when at operation, pus was noted coming from the general abdominal cavity outside the local field. If these criteria were not fulfilled, the peritonitis was classified as "acute local."

AGE INCIDENCE

It has long been known that appendicitis occurs more frequently in young individuals than in older people; but it is by no means limited to the young.

The figures reported from various metropolitan hospitals[4, 5] and from the Mary Imogene Bassett Hospital show (Fig. 2) that after reaching a peak in the second or third decade the number of cases falls off rapidly in the fourth decade. After this point the Bassett Hospital curve flattens out, while the others continue to drop; and in the sixth and seventh decades the divergence is striking. This is, of course, due to the fact already pointed out that a greater proportion of the population here is in an older age group than in most communities, particularly cities. That we are indeed dealing with an older age group than is usual is shown even more strikingly in Figure 3, where the percentage at this hospital of patients over 50 years of age with acute appendicitis is contrasted with similar percentages reported from various metropolitan hospitals. It will be observed that 15.5 per cent of the patients at the Bassett Hospital (taking into consideration the two series of cases) are over 50 years of age, as contrasted with 3.4 per cent at the Boston City Hospital,[6] 5.8 per cent at the Cincinnati General Hospital,[7] and 3.4 per cent at the New York Hospital.[8]

SPECIAL PROBLEMS OF ACUTE APPENDICITIS IN THE OLDER AGE GROUP

Acute appendicitis as seen in elderly patients varies markedly in at least three respects from the disease in adolescents and young adults. The ability of the body to combat infection is decreased in the elderly; the frequency of serious postoperative vascular and pulmonary complications is sharply increased; and diagnosis is often much more difficult, since the classic picture of acute appendicitis is not common in this age group.[9-11] Stalker,[9] in reviewing 82 cases of appendicitis in patients over 60 years of age, found that more than half of them were atypical. Muscle spasm is less marked; and localizing signs may be poor or almost completely absent. Powers[10] has pointed out that an attack of appendicitis in the elderly patient is often ushered in by pain of a diffuse, lower abdominal type, similar to that associated with pathologic conditions of the colon, rather than by the classic epigastric pain, subsequently referred to the right lower quadrant, which is encountered in younger individuals. Furthermore, the differential diagnosis when the patient is of the older age group must take into consideration various diseases not common in younger patients, such as neoplasms of the bowel or diverticulitis with possible perforation.

Gangrene or Perforation. Since the percentage of our patients beyond the age of 50 is approximately three times that reported from other clinics, it would appear reasonable to assume that the disease seen here is correspondingly more serious. Among the 540 cases comprising this report, gangrene or perforation of the appendix, or both, was found in 208 instances or 38.5 per cent of the cases. It will be noted in Figure 4, showing the age distribution of these complications, that the incidence is low in the second, third and fourth decades, and that the curve rises sharply in the succeeding decades. It is also interesting to note that the incidence is high in the first decade.

DURATION OF SYMPTOMS PRIOR TO ADMISSION

The promptness with which patients entered the hospital following the onset of symptoms is shown on Figure 5. Fifty-eight per cent of the patients had been admitted within the first 24 hours; 82 per cent within 48 hours. For comparison with urban centers, figures reported from the New York Hospital[8] for the years 1932–1937 have also been charted in Figure 5, and it will be observed that patients enter the hospital here rather more promptly than they do in the city. At first glance this may seem surprising, since many of the patients in this area live at a considerable distance from the hospital; but on reflection it is obvious that generally speaking, two-thirds of the patients with

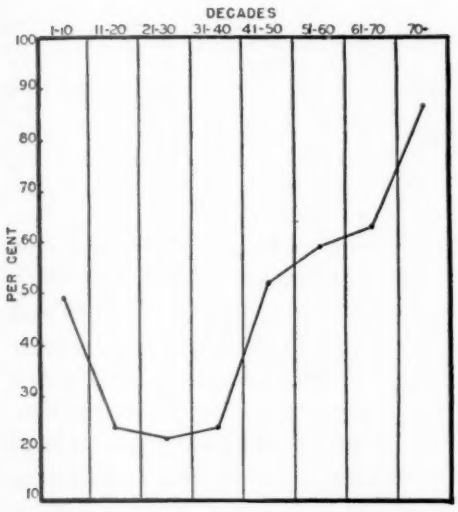

FIG. 4

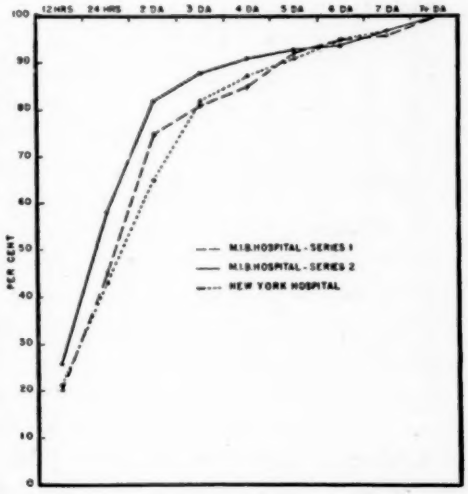

FIG. 5

FIG. 4.—Age incidence of gangrene or perforation or both, occurring in patients with acute appendicitis at the M. I. B. Hospital.

FIG. 5.—Percentage of patients with acute appendicitis admitted to the M. I. B. Hospital (Series I and II) and to the New York Hospital at various time intervals after the onset of symptoms. It will be noted that the figures from this rural district compare favorably with those from the metropolitan area; and, further, that the promptness of hospitalization has improved in this community through the past ten years.

acute appendicitis, whether here or in New York, wait 12 hours or more before entering the hospital, and that the extra time which patients in a rural district may have to spend in transit is of little importance in the total period of time elapsing between onset of symptoms and hospitalization. The somewhat greater speed with which patients enter the Bassett Hospital may be due in part to the fact that in a rural area it is often possible to have much better liaison with the referring physicians, which doubtless contributes to the speed with which arrangements can be made; and may be partly because

a larger proportion of the patients present themselves directly at the hospital, thus eliminating one step in the process of admission.

It is worth noting, in connection with the general effort to educate the public along medical lines, that the patients of the second series (1938–1949) reached the hospital sooner than those of the first series (1930–1938) (see Fig. 5). It would be interesting to compare the figures of the second series with those of a late series from New York city. A recent report from the Cincinnati General Hospital[12] shows that as a result of intensive public education in that city there has been marked improvement in the promptness of admission.

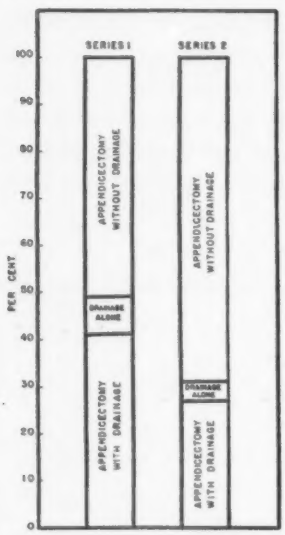

FIG. 6. — Operative procedures in the first and second series of patients with acute appendicitis at the M. I. B. Hospital.

OPERATIVE PROCEDURES

Operative procedures at the Bassett Hospital during the years covered by this report have been carried out by the same two senior surgeons and a number of different members of the house staff. In the great majority of instances the classic McBurney incision has been used, although the low right rectus muscle-splitting approach has been employed when it was thought to be indicated. Our feeling as to the proper treatment of the appendiceal stump is essentially that of Holman,[13] who believes that in no form of intestinal surgery should a mucosa-to-mucosa closure be considered adequate and that the "tie and drop" method should not be employed. In our cases, therefore, the appendiceal stump has routinely been ligated and buried, using a purse-string suture.

There has been a rather marked change between our two series in the use of drainage. Prior to 1938, appendicectomy with drainage was carried out in 41 per cent of our cases, and drainage alone in 8 per cent (Fig. 6). Since 1938, the percentage of appendicectomy with drainage has been reduced to 27, and that of drainage alone to 4. This change is in large part due to the influence of antibiotic therapy.

MORTALITY

There were 17 deaths in the total 540 cases, or an over-all mortality of 3.1 per cent. In the first series of 182 cases there were eight deaths, as shown in Table II, or a mortality of 4.4 per cent; in the second series there were but nine deaths in 358 cases, a mortality of 2.5 per cent. It is of interest to note that 44 per cent of the deaths occurred in the 15.5 per cent of the patients over 50 years of age.

In the last three and a half years, during which time 123 patients with acute appendicitis were operated upon at this hospital, there has been but

one death—that of a 13-year-old boy who succumbed to typical ether convulsions—giving a mortality rate for that period of 0.8 per cent.

TABLE II.—*Mortality.*

	Number of Cases	Number of Deaths	Mortality Per Cent
Series I	182	8	4.4
Series II	358	9	2.5
Total	540	17	3.1

It is of further interest to break down the mortality statistics in relation to the pathology encountered at operation (see Table III). In the 314 cases of simple acute appendicitis there was but one death—mentioned above—a mortality in this group of 0.3 per cent. Among the 91 cases with acute local peritonitis, there were three fatalities, or a mortality of 3.3 per cent; of the 53 patients with peritoneal abscess, three died, a mortality of 6.0 per cent; and among the 82 patients with acute diffuse peritonitis there were ten deaths, bringing the mortality in this group to 12 per cent. It has already been stated that 208 of the patients had perforation or gangrene of the appendix; among these patients the mortality was 7.7 per cent.

TABLE III.—*Pathology Found at Operation 1930–1949.*

Pathology	Number of Cases	Number of Deaths
Simple appendicitis	314	1 (.3%)
Acute diffuse peritonitis	82	10 (12%)
Acute local peritonitis	91	3 (3.3%)
Peritoneal abscess	53	3 (6%)
Total	540	17 (3%)

Appendix stated to be perforated or gangrenous in 208 cases, or 38.5% of the total number.

As shown by Table IV, the most common cause of death was acute diffuse peritonitis, which was responsible for nine of the total 17 deaths. Pulmonary embolus was listed as the cause of death in five instances; while wound infection, convulsions during anesthesia, or cerebral hemorrhage was responsible for the remaining three deaths.

Discussion. The present report, in common with similar reports from representative large clinics,[3, 12, 14] shows a gratifying decline in the mortality from acute appendicitis. Several factors are undoubtedly partly responsible for the falling death rate. Surgeons are on the whole better trained, and surgical house staffs are better organized and supervised. The general public is better educated in the importance of reporting to a doctor promptly in case of acute abdominal pain; and is also more aware of the hazard of taking a cathartic under such circumstances.

But other factors are of even more significance in the reduced mortality. Advances have been made in the preoperative and postoperative care of patients, notably through a better understanding of fluid, electrolyte and protein requirements; through the use of the Miller-Abbott tube for decompression of the intestine; and, of more importance, through the introduction of the antibiotics and the anticoagulant drugs. The effect of these two latter discoveries on the mortality in cases of acute appendicitis at the Bassett

TABLE IV.—*Cause of Death.*

Cause of Death	Number of Cases
Acute diffuse peritonitis	9
Pulmonary embolus	5
Spreading wound infection (with bronchopneumonia)	1
Convulsions during anesthesia	1
Cerebral hemorrhage	1
Total	17

Hospital is striking. It has been noted in Table IV that the principal cause of death, when the total group of cases is considered, was acute diffuse peritonitis; but there has been no death from peritonitis since 1945, a date which corresponds roughly with the first use of penicillin in this hospital. As also noted in Table IV, the second most frequent cause of death was pulmonary embolism; and from this cause we have had no deaths in cases of acute appendicitis since 1940. The use of anticoagulant drugs has been an important factor in this improvement, but by no means the only one. Ligation of the superficial femoral veins has been carried out in appropriate cases since 1938. Early ambulation[15] was started in this hospital in 1940, and while this has not prevented the occurrence of thrombophlebitis it has definitely lowered the incidence of fatal pulmonary embolism.[15] Within the last few years we have done very few vein ligations, placing our reliance almost entirely on anticoagulant drugs. These have been used not only when there was evidence or suspicion of thrombophlebitis, but also as a prophylactic measure for patients of the type shown by experience to be particularly likely to suffer a fatal embolus: notably the elderly, obese individuals with peritonitis or an intraperitoneal abscess.

SUMMARY

1. Two series of patients with acute appendicitis operated upon at the Mary Imogene Bassett Hospital are reported.

2. In this rural area there is an unusually high percentage of patients in the older age groups. The special problems presented by elderly patients with acute appendicitis include difficulties of diagnosis, relatively high incidence of gangrene and perforation, and frequency of serious postoperative vascular and pulmonary complications.

3. The interval of time between onset of symptoms and admission to this hospital in the two series compares favorably with that reported from various metropolitan hospitals. The considerable distance from the hospital of many of the rural patients appears to be a negligible factor in this interval. It is noteworthy that patients of the second series entered the hospital more promptly than did those of the first series, reflecting better education of the public in regard to medical matters.

4. The mortality in the second series (2.5 per cent) is markedly lower than in the first series (4.4 per cent). This is due largely to the introduction of antibiotics, which has drastically reduced the mortality from acute diffuse peritonitis; and to the use of various prophylactic measures against pulmonary embolism, including ligation of veins, early ambulation, and the use of anticoagulant drugs. There has been no death from peritonitis in cases of acute appendicitis since 1945, and none from pulmonary embolism since 1940.

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PYLORIC HYPERTROPHY IN THE ADULT*

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CONGENITAL PYLORIC HYPERTROPHY has long been recognized as a distinct clinical entity in infants. It is not so generally realized that there may occur in adults a lesion which bears a striking resemblance to it. It is important that this disease be differentiated from other lesions at the pylorus because, although often disabling, it is benign and amenable to surgical correction.

Both the infantile and the adult forms of hypertrophy were noted at autopsy many years before they were accorded clinical recognition. The familiar congenital pyloric hypertrophy was first described in 1627 by Fabricius Hildanus and at least three authentic cases were recorded[27] prior to the first report in the American literature by Hezekiah Beardsley[6] in 1788. A similar situation applied to the adult form of pyloric hypertrophy. In the Atlas of Pathological Anatomy, published in 1842 by Jean Cruveilhier,[27] there appeared an illustrative drawing of an enormously dilated stomach with hypertrophy of the pylorus found at autopsy upon a 72-year-old woman who had a history of vomiting throughout her life. It is customary to accord priority to Maier[28] for describing this disease. However, close scrutiny of his paper indicates that he was describing incidental gross findings at autopsy in patients whose clinical histories were either fragmentary or completely lacking. Some of his pyloric measurements were well within normal limits, and no microscopic examinations were recorded. The description of the lesions suggests that many of these patients had infiltrative carcinoma or cicatrizing ulcers.

Secondary pyloric hypertrophy is commonly observed with both benign and malignant ulceration of the stomach. Many authors[23, 24, 37, 47] have called attention to this and have noted that the associated lesion need not necessarily be in the immediate prepyloric region. It is found also in association with gastric polyposis[37] and in hypertrophy of the mucosal rugae, particularly if the hypertrophied mucosa blocks the pyloric orifice.[46] The disease with which we are concerned appears to be a primary hypertrophy of the pyloric muscle since none of these associated gastric abnormalities are present. Mayo-Robson[29] in 1904 reported the first surgical cure. He performed a gastrojejunostomy with a good result in a 24-year-old patient with hypertrophy of the pylorus whose symptoms had recurred after forcible stretching of the muscle. Barling[5] appears to have been the first to report microscopic examination of tissues. Subsequently, numerous authentic cases of pyloric hypertrophy without other significant lesions of the stomach have been reported. We have been able to collect 59 cases with operative or necropsy verification and are adding five previously unreported cases (Table I).

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Tabulation of Authenticated Cases

Author	Year	Age	Sex	Major Symptoms	Duration	Diagnosis Established by	Treatment	Result
Mayo-Robson and Moynihan	1904	24	M	Discomfort in epigastrium	5 yrs.	Operation	Gastro-enterostomy	Well
Barling, No. 1	1913	27	M	Pain and vomiting	3 yrs.	Operation	Gastro-enterostomy	Well
Barling, No. 2	1913	17	F	Pain and vomiting	6 mos.	Autopsy (micros.)	Gastro-enterostomy	Died
Reynolds	1913	35	M	Dyspepsia and vomiting	3 mos.	Autopsy (gross)	Medical	Died
Oliver	1922	51	M	Indigestion and vomiting	Life	Operation	Pyloromyo-tomy	Well
Chaney	1928	25	F	Pain and vomiting	9 mos.	Operation	Pyloroplasty	Well
Crohn	1928	45	F	Vomiting	Life	Operation	Gastro-enterostomy	Survived
Ackman	1929	72	M	Pain and vomiting	Life	Autopsy (micros.)	Medical	Died
Archer	1930	63	M	Distress and vomiting	5 mos.	Operation	Pyloromyo-tomy	Improved
Archer	1930	42	M	Distress and vomiting	6 yrs.	Operation	Dilatation	Recurrence 4 mos.
Archer	1930	39	F	Flatulence and vomiting	1 yr.	Operation and path.	Pyloroplasty	Well
Guiou	1930	35	F	Vomiting	20 yrs.	Operation	Resection	Well
LaPorte and Scholz	1931	48	M	Pain, indigestion and vomiting	8 yrs.	Operation and path.	Resection	Well
McClure, No. 1	1931	55	F	None	?	Autopsy	None	Cardiac death
McClure, No. 2	1931	36	F	Pain and vomiting	Life	Operation and path.	Resection	Well
McClure, No. 3	1931	44	F	Pain	Life	Operation and path.	Resection	Well
McClure, No. 4	1931	45	M	Pain and vomiting	33 yrs.	Operation and path.	Resection	Improved
Coleman	1932	38	M	Pain and vomiting	7 yrs.	Operation and path.	Resection	Well
Stone and Ruggles	1932	46	F	"Waterbrash"	Life	Operation	Pyloromyo-plasty	Well
Wauchope	1932	68	F	Vomiting	53 yrs.	Operation	Pyloroplasty	Well
McNamee	1933	40	M	Pain	10 yrs.	Operation and path.	Resection	Well
McNamee	1933	14	M	Pain and vomiting	4 mos.	Operation	Pyloroplasty	Well
Pendergrass	1933	24	F	Pain and vomiting	Life	Operation	Gastro-enterostomy	Well
Twining	1933	43	F	Pain and vomiting	10 yrs.	Operation and path.	Resection	Survived
Twining	1933	55	M	Anorexia and flatulence	6 mos.	Operation and path.	Surgery	Survived
Twining	1933	50	M	Flatulence	Life	Operation and path.	Resection	Survived
Elmer and Boylan	1934	68	M	Pain, flatulence and vomiting	5 wks.	Operation and path.	Pyloroplasty and gastro-enterostomy	Well
Bockus	1943	68	M	Pain and vomiting	3 yrs.	Operation and path.	Resection	Well
Kestel	1938	55	M	Pain and vomiting	3 mos.	Operation and autopsy	Gastro-enterostomy	Died, 5 mos.
Berk and Dunlap	1944	39	M	Distress and vomiting	5 yrs.	Operation and path.	Resection	Well
Berk and Dunlap	1944	37	M	Fullness and vomiting	3 mos.	Operation and path.	Resection	Well

Tabulation of Authenticated Cases (Continued)

Author	Year	Age	Sex	Major Symptoms	Duration	Diagnosis Established by	Treatment	Result
Wakefield	1944	52	M	Nausea and vomiting	Life	Operation and path.	Resection	Well
Andersen, et al.	1946	61	M	Pain and vomiting	35 yrs.	Operation and path.	Resection	Survived
Andersen, et al.	1946	53	M	Pain	5 yrs.	Operation and path.	Resection	Died—4 days
Vorhaus	1946	46	M	Pain and vomiting	4 yrs.	Operation and path.	Resection	Well
Katz	1947	34	M	Pain and vomiting	7 yrs.	Operation and path.	Resection	Well
Hobson	1948	55	F	Pain, fulness and vomiting	1 yr.	Operation and path.	Resection	Well
Hobson	1948	54	F	Indigestion and vomiting	1½ yrs.	Operation and path.	Resection	Survived
Mazzetti	1948	44	F	Pain, fullness and vomiting	19 yrs.	Operation and path.	Resection	Well
North and Johnson	1949	49	F	Flatulence and vomiting	5 yrs.	Operation and path.	Resection	Died—7 days
North and Johnson	1949	43	M	Pain and vomiting	2 mos.	Operation and path.	Resection	Well
North and Johnson	1949	53	M	Pain and vomiting	4 yrs.	Operation and path.	Resection	Well
North and Johnson	1949	66	M	Nausea—flatulence	6 mos.	Operation and path.	Resection	Well
North and Johnson	1949	85	M	Dysphagia and vomiting	6 mos.	Autopsy (micros.)	Medical	Died

Judd and Thompson in 1933 reported 20 cases from the Mayo Clinic. This series can only be analyzed as follows:

Age	Sex	Major Symptoms	Duration	Diagnosis Established by	Treatment	Result
23 to 64	M—19 F — 1	Vomiting....17 Weight loss..13 Tumor..... 2	Averaged 10½ yrs.	Operation in...20 Path. exam. in.11	Ramstedt..... 4 Pyloroplasty...10 Resection..... 6	Improved.....13 Unimproved..... 2 Died (other causes) 2

ETIOLOGY

It is natural to assume that hypertrophy in adults represents a persistence of the infantile form because morphologically the lesions are almost identical. The clinical history of lifelong indigestion and vomiting given in some of the reported cases also lends support to this concept. If all patients presented this typical history, it would be unnecessary to seek any other etiologic explanation. However, a more common history is that of a more or less abrupt onset of digestive disturbances in a previously healthy, middle-aged adult. Nevertheless, about one-quarter of the cases recorded in the literature[1, 12, 31, 34, 36, 40, 42, 44] had intermittent symptoms of pyloric disturbance persisting throughout infancy and childhood into adult life. The remarkable history presented by Ackman[1] concerns a man who died of starvation at the age of 74, having steadfastly refused operation despite recurrent attacks of severe abdominal colic and vomiting throughout his entire life.

Runstrom[39] had an opportunity to observe over a period of years a large

group of children who had congenital pyloric stenosis. He noted that roentgenologic abnormalities remained long after active symptoms had subsided. During the "manifest" stage, he observed a dilated stomach terminating abruptly in a smooth elongated pyloric canal. Peristaltic waves were interrupted at the constricted portion, and the passage of barium into the duodenum was prolonged from two to four times normal. When these patients were re-examined later in the first year of life, the pyloric canal was wider and the outline of barium within it showed longitudinal streaks. The latter he interpreted as mucosal rugae which had been obliterated by edema during the stage of clinical symptoms. This stage appeared to continue for one to four years. These abnormalities gradually regressed but in some cases a normal pattern was not established even as late as the thirteenth year. These observations by Runstrom show clearly that an organic basis may remain in unoperated patients as a potential source of trouble in adult life.

It must be admitted that the male sex preponderance is not as overwhelming among adult cases as is generally accepted for infantile hypertrophy of the pylorus. In the 64 cases in the collected series, including our own, the ratio of males to females is only 3 to 1. However, in the series of 20 proved cases reported by Judd and Thompson,[20] the largest single group on record, this sex ratio was 19 to 1.

Some[10, 31, 43, 47] have rejected the congenital theory in favor of the view that protracted pylorospasm is responsible for the muscular hypertrophy of the sphincter. This hypothesis is not well supported by analogy. For example, anal fissure is associated with severe and protracted spasm, yet no hypertrophy of the anal sphincter muscle is apparent in this condition. Nor is there observable thickening of the sphincter of Oddi in the cases of biliary disturbance attributed to ampullary spasm. Moreover, if it were true that spasm in itself produces hypertrophy of a sphincter, then the radiologist who encounters pylorospasm almost daily in his fluoroscopic observations should likewise frequently discover pyloric hypertrophy.

Boerema's account[9] of gastro-intestinal diseases observed in the Netherlands during the Nazi occupation is an interesting sidelight on this spastic theory. He observed that the restricted diet, mainly coarse carbohydrate foods, upon which these people subsisted, gave rise to various spastic diseases of the gastro-intestinal tract. During this period Dutch roentgenologists observed "many instances" of a rigid gastric antrum in which hypertrophy of the pylorus, without ulcer or cancer, was the sole finding at operation. It is unlikely that malnutrition accounted for this finding. Atony rather than hypertrophy is customary in people dying of starvation. We have searched in vain for reports of pyloric hypertrophy in the studies made upon our liberated prisoners of war; but, apparently, no gastro-intestinal roentgenograms were done on these men. Our interest in malnutrition as an etiologic factor was aroused by obvious deficiencies in the dietary habits of two of our patients, but the report of Boerema is the only evidence to support such a theory.

Summation of the evidence upon the etiology of this lesion leads to the conclusion that probably a thickening of the pyloric muscle persists asymptomatically from infancy in these patients. This is the basic abnormality and sometime in adult life a complicating factor may occur to precipitate the symptoms of pyloric occlusion. This factor may be inflammatory thickening, the edema of electrolyte imbalance or spasm.

CLINICAL MANIFESTATIONS

There is no characteristic clinical picture of pyloric hypertrophy. It has been noted that commonly the first symptoms do not appear until adult life. In 70 per cent of the reported cases the onset was between the ages of 31 and 60. In one of our cases it was delayed until the ninth decade. The manner in which the symptoms appear also varies. Sometimes there are mild, intermittent manifestations which gradually become more severe and constant; in others complete pyloric obstruction may develop without prior warning. If one assumes that simple hypertrophy of the pylorus may not interfere with function unless it is sufficient to produce occlusion, these variations in symptomatology are understandable. In about 70 per cent of the reported cases epigastric pain or discomfort and vomiting are the presenting symptoms. The pain varies in intensity but usually is intermittent, aggravated by taking food and not susceptible to relief by antacids. In some cases it is so mild that it is described only as a sense of fullness after eating. Rather uniformly, vomiting relieves the discomfort. In patients with a long standing history of vomiting, nutritional deficiencies and weight loss occur, and indeed are a significant feature in about half the reported cases. There is a small group of patients who have pain without vomiting and also a few who vomit without any accompanying distress. A palpable abdominal mass was found in only four cases. It is obvious that neither history nor physical findings contribute significantly to the diagnosis of pyloric hypertrophy.

ROENTGENOLOGIC FEATURES

The roentgenologist is the one who most frequently discovers the condition, although more often than not he can not distinguish it from an infiltrative neoplasm. Ordinarily, it is not confused with benign ulceration. Delay in the emptying of the stomach is usually observed fluoroscopically but in contradistinction to annular carcinoma or stenosing ulcer, it is rare to find complete gastric retention. Characteristically there is a 25 to 50 per cent retention at the end of six hours. Extreme degrees of hyperperistalsis or gastric dilatation are unusual. Due to the fact that a portion of the barium meal enters the duodenal bulb, the pyloric canal can be visualized and shows significant elongation. The narrow passage through this canal may be either concentric or eccentric, depending upon whether or not the muscular hypertrophy is uniformly distributed. When careful attention is given to the mucosal pattern, irregularities produced by rigid folds of edematous mucosa

may sometimes be observed projecting into the barium column. They may be distinguished from those of a pyloric ulcer by the fact that flecks of barium are not retained. The abnormalities described are persistent after the administration of antispasmodic drugs. Kirklin[23] regards a mushroom or umbrella-shaped deformity at the base of the duodenal bulb as an almost pathognomonic sign of pyloric hypertrophy and on this basis reported a large series of cases, most of which were not verified. This roentgenologic sign was described by Pendergrass[15, 35] as indicating prolapse of gastric mucosa or of pedunculated tumors. It may be present both in pyloric hypertrophy and in prolapse but in the latter the defect may be varied by palpation which alters the position of the intussuscepted gastric mucosa or tumor. The same defect in the base of the duodenal bulb may also be produced by the forward pressure of an accumulation of barium in a dilated gastric antrum proximal to a narrowed pyloric ring. We have observed this in cases of pyloric ulcer. This ballooning effect is unlikely to occur in adults with pyloric hypertrophy because of the rigidity produced by the hypertrophy and the edema of the prepyloric segment in this condition. A mushroom deformity of the base of the duodenal bulb, therefore, is probably not a reliable roentgenologic sign of pyloric hypertrophy.

THE APPEARANCE OF THE LESION

It is not always easy to recognize this lesion at the operating table. A firm tumor mass, or at least a thickening, is usually but not invariably palpable. When the stomach appears normal, the surgeon is obligated to open the prepyloric portion of the stomach and inspect its interior. The pyloric ring should not be divided until after palpation has been done with a finger introduced through the pyloric canal, because once the sphincter is cut and its ends retracted it becomes very difficult to evaluate the situation. If direct examination is negative, the gastrotomy incision may be closed transversely or converted into a pyloroplasty after extending the incision into the duodenum. Many pyloric lesions will be missed unless gastrotomy is made a routine part of surgical exploration.

The normal variations in the thickness of the pyloric muscle have never been clearly defined. Truesdale[41] and Horowitz *et al.*[19] agree that 0.5 cm. is an average measurement, although the range in the latter's series was from 0.38 to 0.85 cm. and both made their measurements upon fixed specimens in which the sphincter had been sectioned. In most of the recorded cases upon which data are available, the pyloric muscle measurements were considerably above this normal range.

The outstanding pathologic feature of this lesion is hypertrophy of the circular layer of muscle in the pyloric ring. The longitudinal muscle coat may also show a moderate grade of hypertrophy and the submucosa and mucosa may be thickened by edema or chronic inflammatory reaction. In the gross specimen one observes an unyielding tumor occupying the distal three or four

centimeters of the stomach. It is thickest at the pyloro-duodenal junction and fades out gradually over the gastric antrum. The muscle hypertrophy stops abruptly at the pyloric ring. Microscopic examination confirms the gross findings. There is hypertrophy and hyperplasia chiefly of the circular muscle of the pylorus, but occasionally involving the longitudinal coat also. In the submucosa and the mucosa there are usually foci of cellular infiltration, consisting of plasma cells, lymphocytes and a few neutrophils. This represents a mild non-specific inflammatory reaction and shows no tendency to proceed to suppuration. The intensity of the inflammatory reaction seems to be proportional to the duration and degree of pyloric obstruction.

TREATMENT

It is obvious that in some cases the symptoms may be so mild and indefinite that no treatment is indicated. It is noteworthy that two patients have lived beyond the age of 70, even though their roentgenologic findings aroused suspicion of pyloric carcinoma and operation was recommended because of this. It is mainly due to the great difficulty in distinguishing this disease from cancer that surgical exploration is advocated. It needs to be re-emphasized that gastrotomy may be necessary to exclude pyloric ulcer, leiomyomata, hypertrophic gastritis, prolapsing mucosa or pedunculated tumor, any of which may be coincidental with or responsible for pyloric hypertrophy. Various types of surgical procedure have given uniformly good results. An exception is simple dilatation of the pylorus, which, in the experience of Mayo-Robson[29] and of Morton,[33] was followed by recurrence of symptoms. Gastro-jejunostomy has generally been satisfactory. It is well to bear in mind, however, that Donovan,[14] in operating upon adults who had been treated surgically for the infantile form of pyloric hypertrophy, noted that the pyloric tumor remains unchanged after gastro-jejunostomy; whereas, a normal appearing pylorus is the rule after a Ramstedt pyloromyotomy. Pyloroplasty corrects the abnormality but has the disadvantage that sufficient tissue may not be obtained for biopsy and thus a neoplasm may be overlooked. By modern surgical standards, a limited gastric resection involves essentially no additional risk and should be the procedure of choice rather than gastro-jejunostomy or pyloroplasty.

One of the authors (J. P. N.) first encountered this disease several years ago while associated with Dr. E. L. Eliason of Philadelphia. More recently, two cases have been seen in the Veterans Hospital, McKinney, Texas. Dr. Eliason has graciously permitted the reporting of this early case and has provided the records of two additional cases subsequently encountered in his practice.

Case 1.—Mrs. A. B. was 49 years old, and ever since girlhood had a vague sort of indigestion. At the age of 44 the symptoms became more definite, in the form of attacks of postprandial discomfort, heartburn and bloating. She at first obtained relief by drinking hot water, but eventually she resorted to inducing vomiting and would often vomit food eaten 5 to 6 hours previously. About 6 weeks before her admission to the University of Pennsylvania Hospital, her symptoms became constant and more severe

and she vomited several times each day. Roentgen ray examination showed an almost complete 6 hour gastric retention. There was a variable defect in the pyloric region and a tendency to a hollowed-out appearance at the base of the duodenal cap. It was suggested that hypertrophied mucosa might explain the rather unusual appearance of the films. The patient was operated upon on December 4, 1929. There was a thickening and a stenosis of the pylorus which was suspected of being carcinoma, and a partial gastrectomy was done. The resected specimen showed symmetrical hypertrophy of the pyloric ring, having the appearance of a presenting cervix when viewed from the duodenal side. There was no ulceration of the mucosa. The thickened muscle merged gradually with the normal coat of the gastric antrum. Microscopic sections showed marked hypertrophy of the muscularis with fibrous trabeculations passing through it and no evidences of malignancy. The pathologic diagnosis was "linitis plastica involving the pyloric end of the stomach."

Unfortunately, this patient died about a week after operation from intestinal obstruction at the site of the anastomosis. Her history of lifelong indigestion supports the view that she had survived to adult life with congenital pyloric hypertrophy.

Case 2.—J. C. D. This 43-year-old laborer could recall no significant digestive complaints until, rather abruptly on the night of November 15, 1947, he developed epigastric pain and vomited a large quantity of undigested food. During the ensuing 5 weeks until his admission to the McKinney Veterans Hospital he vomited daily and lost 34 pounds in weight. For many years his economic status was such that his diet had been seriously deficient in protein. The physical examination showed nothing abnormal other than evidence of poor nutrition. He weighed 108 pounds.

The erythrocyte count was 4,400,000 with 13 Gm. of hemoglobin. No blood was found in repeated stool examinations. The gastric analysis showed free hydrochloric acid ranging from 6 to 11 units and total acids from 18 to 38. No tumor cells were found in the gastric washings.

Roentgen ray examination of the gastro-intestinal tract revealed a narrowing of the distal third of the stomach and no peristaltic waves passed through this area. After 24 hours about one-third of the barium meal was still retained in the stomach. The margins of the defect in the distal part of the stomach were irregular, suggesting destruction of the mucosa. A second examination made after the patient had received atropine to the point of tolerance showed the same abnormalities. The impression of the radiologist was that this was an annular carcinoma of the distal stomach (Fig. 1).

On this assumption the patient was prepared for laparotomy by correction of his electrolyte imbalance and tube drainage of his stomach. Laparotomy was performed on January 26 under continuous spinal anesthesia through a right transverse incision. There was some edema of the wall of the stomach and a firm, almost cartilaginous, annular lesion involving the pyloric ring and about 3 cm. of the prepyloric region. The stomach was opened to visualize the pylorus, which was found to be almost completely occluded. This was suspected of being an infiltrative neoplasm. The distal two-thirds of the stomach were resected with a wide margin of normal stomach wall. The duodenal stump was inverted and a retrocolic Hoffmeister type of end-to-side gastrojejunostomy performed. On the seventh day he was taking a soft diet without difficulty and he was discharged on the twenty-first postoperative day.

The pathologist found 3 soft lymph nodes along the great curvature. The pylorus measured 3 cm. in circumference and its lumen had a diameter of about 1 cm. The pyloric mucosa which was intact was not fixed to the underlying muscularis and showed a complete absence of rugae. The thickness of the wall of the stomach varied from 2.0 cm. in the pyloric canal to 1.0 cm. at the point where the hypertrophy merged with a normal gastric wall (Fig. 2).

Microscopically, a section through the pylorus showed a normal intact mucosa, a markedly edematous submucosa and an edematous and hypertrophic muscularis. The muscular layer showed hypertrophy of the individual cells and the muscular bundles were separated by edema and by many foci of chronic inflammatory cells, including lymphocytes, plasma cells and a few polymorphonuclear cells. There was nothing to indicate a suppurative or neoplastic process either in the stomach or in the regional lymphnodes. An occasional arteriole in the submucosa showed thickening of the media and subintimal hyalinization. The chronic inflammatory foci were not consistently perivascular in distribution. The pathologic diagnosis was hypertrophic pyloric stenosis with chronic inflammation.

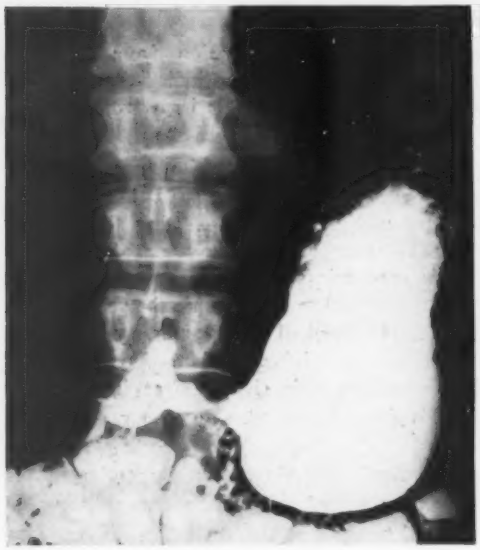

FIG. 1

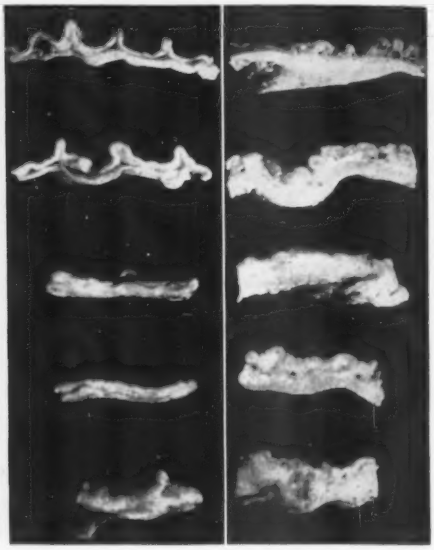

FIG. 2

FIG. 1.—Roentgenogram of stomach in Case 2 showing persistent narrowing and elongation of pyloric canal. About one-third of the barium meal was retained in the stomach after 24 hours.

FIG. 2.—The gross thickening of the pyloric and prepyloric portions of the stomach wall in pyloric hypertrophy is shown by this comparison between a normal stomach (left) and that of Case 2 (right). The lowermost section in each column is taken at the pyloric ring. Sections above are taken at corresponding levels through the prepyloric region in the two stomachs. It is evident that the hypertrophy at the pylorus is about twice the normal thickness and that this thickening extends proximally for 2 to 3 cm.

Case 3.—L. A., a 53-year-old male, came to the McKinney Veterans Hospital because of periodic peri-umbilical cramps for the past 3 to 4 years. These episodes occurred without warning and bore no relation to meals, but would continue for from 3 days to a week. He obtained some relief by lying down or by drinking milk. For 2 years he had voluntarily restricted his diet to milk, bread, eggs, boiled potatoes and cereals, since other foods increased his distress. On this restricted diet he had lost 10 pounds in the preceding 2 months. He was frequently nauseated during his attacks but only rarely vomited. There were no abnormalities on examination except for evidence of poor nutrition and of poor oral hygiene.

The red blood cell determination was 5,400,000 with 15 Gm. of hemoglobin. Four stool examinations were negative for blood. The gastric analysis showed 28 units of free hydrochloric acid and a total acid of 48 units.

Two gastro-intestinal roentgen ray studies were made one week apart, the second after atropinization. The findings were similar on both occasions. The prepyloric portion of the stomach was constricted and deformed and peristaltic waves faded out upon reaching this area. The duodenal bulb appeared normal. After 5 hours there was approximately 30 per cent of the barium meal remaining in the stomach. The roentgenologic impression was "a prepyloric annular lesion, probably neoplastic in nature" (Fig. 3).

FIG. 3 FIG. 4

FIG. 3.—Roentgen ray of stomach of Case 3 showing narrowing of pyloric canal which persists after atropinization. There was approximately 30 per cent gastric retention at the end of five hours.

FIG. 4.—The resected portion of the stomach in Case 3. The hypertrophied pyloric ring appears as a light area in the lower part of the specimen.

Laparotomy was performed on March 29, 1948, under general, endotracheal anesthesia through a left transverse incision. Casual examination of the stomach did not indicate any obvious abnormality, but there was a suggestive thickening at the pyloric ring. The stomach was opened to inspect the pylorus more carefully. The tip of a hemostat could be passed through the canal, but it would not admit the surgeon's finger. There was a thick mass of muscle in the anterior margin of the pyloric ring which was thought to represent either an encapsulated leiomyoma or pyloric hypertrophy. The distal third of the stomach was resected and a posterior gastro-jejunostomy of Polya type done to restore continuity. He took a soft diet on the sixth day and was dis-

charged on the eleventh postoperative day. Eleven months later he was having no abdominal pain or vomiting, but he had failed to gain more than a few pounds of weight.

The pathologist described the stomach as having in the anterior wall of the pyloric canal a submucous tumor mass, measuring 3 by 1.5 by 1.2 cm. Although grossly this showed a resemblance to a leiomyoma, it was evident microscopically that the tumorous area merged with a hypertrophy of the entire circumference of the circular coat of the muscularis propria. The asymmetry of the pyloric ring was produced by localized hypertrophy of the muscularis mucosa. Throughout the hypertrophic areas there were foci of non-specific inflammatory reaction characterized by lymphocytes, macrophages, plasma cells and a few neutrophils. The pathologic diagnosis was hypertrophic pyloric stenosis with chronic inflammation (Figs. 4 and 5).

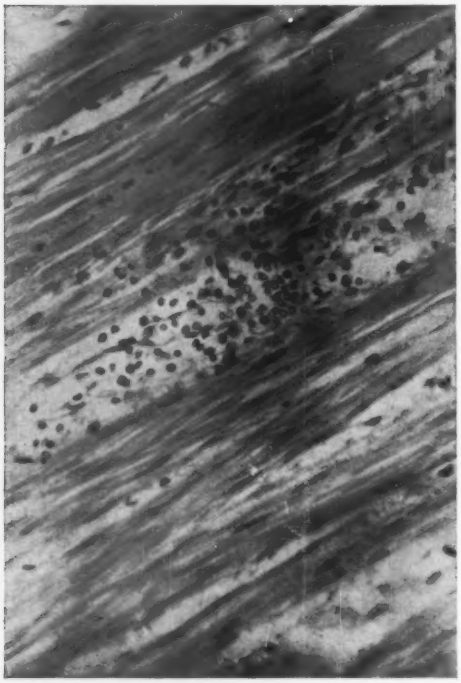

FIG. 5.—Photomicrograph of tissue from pyloric region of Case 3 under high magnification, showing edema and round cell infiltration in addition to the hypertrophy of the muscularis propria.

Case 4.—Mr. R. C. was 66 years old and had had no digestive complaints until approximately six months prior to his admission to the Hospital of the University of Pennsylvania on April 12, 1945. In recent months he had experienced discomfort and nausea after meals. His appetite remained good but he did not eat large amounts because of the distress which they occasioned. He had no definite pain and no vomiting. Over a period of 4 to 5 years he had lost 10 pounds in weight. Gastro-intestinal studies were made by two different roentgenologists. Both agreed in finding an irregular narrowing in the prepyloric region and also a filling defect at the base of the duodenal cap. There was no delay in emptying of the stomach and no retention of barium. One roentgenologist interpreted the findings as probably due to carcinoma, the other favored benign ulcer. One commented that the defect in the base of the bulb might represent an associated hypertrophy of the pyloric sphincter. The patient was operated upon with a clinical diagnosis of pyloric obstruction of uncertain cause. The only abnormality found was a thickening of the gastric wall in the pyloric and antral regions. There was no dilatation of the stomach and no evidence of recent or old ulceration. The surgeon was suspicious that this might be an early lymphosarcoma and performed a partial gastric resection including 14 cm. of the lesser curvature. This afforded complete relief of symptoms.

The pathologist noted no gross abnormalities except for apparent thickening of the pyloric ring, which measured 1.5 cm. in length. The mucosa was intact. Microscopic sections of the pyloric wall showed a great increase in the thickness of the muscular layer and hypertrophy of the individual cells. There was practically no admixture of fibrous tissue. There were a few infiltrating inflammatory cells. In the body of the stomach there was moderate muscular hypertrophy. Pathologic diagnosis was hypertrophy of the pylorus.

Case 5.—Mr. W. H., age 85, was admitted to the Philadelphia General Hospital on October 10, 1946. The details of his clinical history are fragmentary but apparently he had been well until 2 weeks previously. He then began having difficulty in swallowing, and for several days had been unable to take anything except water, but he had no pain or vomiting. On the day of his admission he vomited for the first time with a small admixture of blood in the vomitus. He estimated that he had lost 10 pounds during the 2 weeks of his illness and presented the picture of malnutrition, hypoproteinemia and dehydration. The gastric analysis with histamine showed no free hydrochloric acid and a total acid of only 21 units. A gastro-intestinal roentgen ray study showed normal swallowing function and no abnormalities except at the pyloric region. The stomach was of the cascade variety; peristaltic waves were normal and there was no retention of barium at the end of 3 hours. The pyloric canal was constantly narrowed and elongated—measuring approximately 1.5 to 2.0 cm. in length. Under the fluoroscope there was no demonstrable alteration of the mucosal pattern in the pyloric region but several serial films showed a smooth indentation along the greater curvature aspect of the pyloric canal. Despite some variability in the appearance of this pyloric defect, neoplasm was regarded as the most likely diagnosis.

This patient had multiple senile diseases and operation was postponed. During the period of observation, he developed pneumonia and died.

At autopsy (March 16, 1947) there was found a firm thick pylorus which histologically appeared to be simple hypertrophy of the pyloric muscle without evidence of neoplasm or ulceration. This finding was incidental to the lobar pneumonia and upper urinary tract complications of prostatic hypertrophy which caused his death. This patient is the oldest recorded case of simple pyloric hypertrophy.

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Note: Case 2 remained symptom-free two years after operation.

PARTIAL GASTRECTOMY FOR PEPTIC ULCER

WITH PARTICULAR REFERENCE TO THE PYLORIC ANTRUM*

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AFTER A SERIES of trials extending over half a century, most surgeons presently accept partial gastrectomy as the best of the many operations designed for the treatment of peptic ulcer. They feel it is the safest procedure that effectively reduces the hydrochloric acid content of the stomach and, thereby, the tendency to recurrent ulceration. There is, however, still some discussion as to how the operation should be designed so as to combine maximum effectiveness with minimal risk and mutilation. A solution of this problem depends upon a more exact knowledge of pre- and postoperative gastric physiology.

There can be little doubt that gastrectomy is effective, partly because of the dilution and neutralization of gastric acids, particularly by the secretions of the pancreas and duodenum. Resection of some of the acid-secreting cells of the body also plays a part. Lewisohn,[49] Lahey,[45] Wangensteen[83] and many others feel that it is a very important part and advise resection of a major portion of the stomach. On the other hand, Hunt,[37] Heuer,[30] and Rienhoff[67] are among those who believe that hemi-gastrectomy is usually adequate and is less mutilating. The theory that the pyloric mucosa elaborates a hormone which stimulates the secretion of acid by the fundus is well supported by clinical observations, yet Vale[78] and Gardner and Hart[25] noted no unfavorable results which could be attributed to failure to resect the entire pylorus.

With these problems in mind we have reviewed our own cases.

HALLORAN HOSPITAL SERIES

Between January, 1947, and March, 1949, 101 partial gastrectomies for ulcer were done at Halloran Hospital. There were 70 duodenal, 28 gastric and 3 gastrojejunal or gastrojejunocolic lesions. There was one death. It occurred on the seventy-fifth day following resection for severe bleeding in a 26-year-old man. Autopsy revealed a plastic peritonitis secondary to a leaking duodenal stump and a large anastomotic ulcer. The patient also had carcinoma of the tail of the pancreas with advanced liver metastases. A low resection had been done because of the carcinomatosis and was probably responsible for the anastomotic ulcer. A second case developed a duodenal fistula and subphrenic abscess following exclusion of the antrum with excision

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of the mucosa. The patient eventually made a good recovery. These cases re-emphasize the dangers presented by an edematous and indurated duodenal stump.

The postoperative acid values were studied in 83 cases. In all but four of these, preoperative acids were available for comparison. These values varied considerably in the same patient, especially in the postoperative period. Nevertheless, certain conclusions seem warranted: (a) almost all gastric ulcer

TABLE I.—*Factors Influencing Postoperative Acidity.*

Postop. Acid After Histamine	Cases No.	Cases %	Preop. Acid Over 60	Preop. Acid Under 60	Anastomosis Polya	Anastomosis Hoffmeister	Site of Lesion Gastric	Site of Lesion Duodenal	Pylorectomy Complete	Pylorectomy Incomplete
0	33	40%	12	18	13	19*	16	17	29	4
1–20	22	26%	13	9	10	12	7	15	18	4
21–40	14	17%	12	2	5	9	1	13	13	1
41–60	10	12%	10	0	3	7	1	9	8	2
Over 60 or Anastomotic Ulcer	4	5%	3	0	1	3	0	4	3	1

* Also one sleeve resection.

cases and about 60 per cent of the duodenal ulcers had low postoperative acids (less than 20 units of free acid after histamine); (b) postoperative anacidity occurred most often in cases with low preoperative acids and least often in cases with high preoperative acids; (c) as a group gastric ulcer cases had lower acids both before and after surgery; (d) when the preoperative acid was high (over 60 units of free acid after histamine) resection of 60 per cent or more of the gastric curvature was required to produce

TABLES II and III.—*Height of Resection Correlated with Postoperative Acid.*

	Gastric Ulcers Acid Value p Histamine					Duodenal Ulcers Acid After Histamine			
Resection	0	1–20	21–50	50	Resection	0	1–20	21–50	50
High	2	1	0	0	High	4	5	3	0
Moderate	3	2	1	0	Moderate	9	6	9	1
Low	6	2	0	0	Low	1	1	3	5

anacidity; (e) when the preoperative acid was low, anacidity could be produced in some cases by resection of less than 60 per cent of the curvatures. In our cases, there was no significant difference between the Hoffmeister and Polya anastomoses. It so happened that a majority of the cases with incomplete pylorectomy appeared in the low acid groups postoperatively, despite the fact that resection had been done for duodenal ulcer.

These conclusions are based on the data shown in Tables I, II, and III and in Figure 1. The distal end of the specimen was studied microscopically in all cases and pylorectomy was assumed to be complete only in the 85 cases in which duodenal glands were proved to be present. Tables II and III are

based on roentgen ray studies of the barium filled stomach. We used only films taken under comparable circumstances in an effort to minimize those variations occurring with changes in the position of the tube or patient, with the presence or absence of obstruction, and with variations in the amount of barium ingested. The total linear distance of both curvatures was measured on tracings of the films and the pre- and postoperative measurements for each patient were compared. A resection was called "high" if 70 per cent or more of the linear distance had been resected, "moderate" if between 60 and 70 per cent and "low" if less than 60 per cent.

These observations correspond to the general experience with one important exception, namely the absence of any untoward effect from failure completely to resect the pylorus. Similarly, follow-up observations to date have not shown any such effect. Admittedly, our series is a recent one, but 80 cases, 12 of which had incomplete pylorectomy, have been followed for from nine to thirty months. Other observers have noted that a considerable number of recurrences occur during the first year and, often, a majority of them during the first two years.[3, 40] In the same length of time we have had two cases with definite gastrojejunal ulcer. The autopsy findings in one of them have already been described. Three other patients may have had recurrent ulceration. Unfortunately, they were treated at other hospitals. Two were operated on for peritonitis, but definite perforated ulcer was not found; the other had an episode of recurrent bleeding, but roentgenograms did not show an ulcer. All five of these patients had complete pylorectomy proved microscopically. Four of them had low resections, no more than 50 per cent of the stomach having been removed.

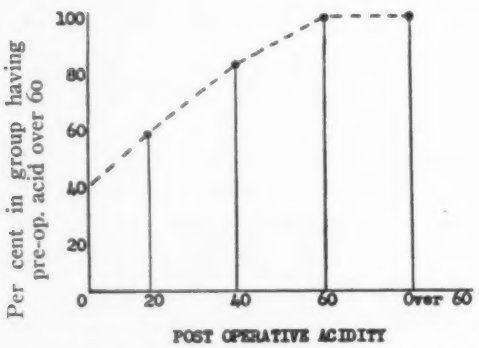

FIG. 1.—Relation between pre- and postoperative acid values.

In brief, these studies again emphasize the necessity of doing a reasonably high resection, especially in the face of high preoperative acids. To date we have been more impressed with the danger of dissecting an edematous pyloric stump than with the harmful effects of leaving it *in situ*. Others have had the same experience.[25, 78] General opinion, however, is so strongly to the contrary that a brief review of the literature seems warranted.

THE RISK OF ROUTINE PYLORECTOMY

Although the mortality rate for gastrectomy has fallen sharply in recent years, the procedure still carries a definite surgical risk. The danger results chiefly from inflammation and contraction of the pylorus and proximal duodenum. As late as 1946 Lahey, in reporting a series of 530 cases, warned

that the "mortality of duodenal ulcer is not that of subtotal gastrectomy but of the relation of the duodenal ulcer to the head of the pancreas . . . and to the common bile duct."[45] This was his explanation of his experience of greater mortality for duodenal than for gastric or gastrojejunal ulcer. St. John,[69] Allen,[5] Wangensteen[80] and many others agree that complete distal

TABLE IV.—*Fatal Surgical Complications After Gastrectomy, Reported from 1941–1948. Non-fatal Complication Listed if Mortality Less Than 0.5 Per Cent.*

			Fatal Surgical Complications							
Authors	Year	No. Cases	Duod. Leak	Perito-nitis	Ab-scess	Bile Duct Injury	Vasc. Acc.	Other	Total	Mortal. %
Colp, *et al.*[19]	1948	100	(5)*	..	(2)*	..	..	..	0	0
Kennedy, Reynolds[39]	1946	90	..	..	..	..	..	..	0	0
King[41]	1946	75	..	..	..	..	..	..	0	0
Sanders[70]	1945	101	..	..	..	..	..	..	0	0
Nissen[62]	1945	220	(2)*	..	..	..	..	1	1	0.4
Garlock[26]	1949	187	1(3)*	..	..	..	..	..	1	0.5
Steinberg[73]	1947	373	..	2	..	..	1	..	3	0.8
Miller, Nicholson[57]	1946	113	..	..	1	..	..	..	1	0.9
Halloran Hospital	1949	101	1	..	..	..	..	..	1	1.0
Rienhoff[67]	1945	260	1	..	..	..	1	1	3	1.1
Mayo Clinic[63]	1942	303	4	..	..	..	..	..	4	1.3
Brooks, Meacham[14]	1945	57	..	..	..	..	..	1	1	1.8
Bradshaw and Hightower[13]	1947	53	..	..	..	..	..	1	1	1.9
St. John, *et al.*[69]	1948	394	5	..	..	..	3	..	8	2.0
Allen and Welch[5]	1946	195	1	3	..	..	..	..	4	2.0
Miller[56]	1942	230	3	..	..	1	..	1	5	2.2
Lahey[43]	1946	530	not itemized: may include some "medical"						12	2.3
Colp, *et al.*[18]	1944									
	'37–40	79	2	..	2	..	..	..	4	5.0
	'40–43	94	1	..	..	..	..	..	1	1.1
Ransom[66]	1947	190	..	3	..	..	..	3	6	3.2
Heuer[30]	1944	142	2	..	..	1	..	2	5	3.5
Cline[16]	1943	83	3	("technical errors")			..	..	3	3.6
Strug[74]	1943	27	..	..	..	..	..	1	1	3.7
Hinton[33]	1943	102	..	4	..	..	..	..	4	3.9
McKittrick, *et al.*[55]	1944	124	3	2	..	..	..	..	5	4.0
Gardner and Hart[23]	1948	123	2	..	..	2	1	..	5	4.1
Bartels and Dulin[11]	1947	100	1	2	..	2	..	1	6	6.0
Allen and Welch[3]	1942	151	1	4	..	2	4	..	11	7.3
Vale[78]	1942	44	2	..	..	1	..	1	4	9.1
Thompson, Prout[76]	1947	41	6	..	..	1	..	..	7	17.0
Larson[47]	1941	116	3	8	1	..	3	6	21	18.1
Totals		4,798							128	2.7

* Not fatal.

resection of the stomach is too hazardous in many cases. We cannot agree with Hinton[33] who stated that every ulcer should be removed while reporting four deaths due to peritonitis in a series of 102 cases. Similarly, Bartels and Dulin[11] state that "irremovable ulcers are not common" but report six surgical deaths in 100 cases. Table IV summarizes the collected experience reported during the past eight years. The total of 128 surgical deaths is not insig-

nificant. "Medical" deaths due to pneumonia, embolism, heart failure, etc., are not included in the table. It should be noted that the large majority of these cases were reported from teaching centers and specialized clinics. An effort was made to ascertain the results in smaller hospitals by reviewing the various state medical journals.[85] Very few of these reports contained mortality statistics. In 1947 Thompson and Prout[76] reported seven surgical deaths in 41 recent resections for ulcer done at a charity hospital.

EXPERIMENTAL

Since the fluid secreted by the pylorus and antrum is usually alkaline in reaction,[63] what is the rationale for routine complete pylorectomy? In 1905 Edkins stated that he had extracted from the pyloric mucosa a specific substance which, when injected into dogs, stimulated the secretion of acid by the cells of the fundus. He called this substance gastrin. This work is still frequently quoted in the surgical literature, although many subsequent experiments appear to have disproved it. The work of Ivy,[38] Priestley and Mann,[64] Wangensteen,[79] Gavin and McHenry[27] and others indicates that the substance extracted by Edkins is probably histamine and that the pylorus contains only about one-fourth as much histamine as the fundus. Best and Taylor[12] believe that "the suggestion that the active principle in pyloric extracts is histamine has been amply substantiated." Histamine can be extracted from many organs outside the gastro-intestinal tract.

On the other hand, there is some reason to suppose that a gastric hormonal or secretagogue substance does exist, although experimenters have as yet been unable to isolate it or to identify its source. Ivy[38] and Klein[42] studied the secretory activity in gastric pouches transplanted to the subcutaneous tissues. They found increased activity in the pouches at variable intervals after feeding. Efforts to show that this secretory stimulant is derived from the pylorus have, in general, been unsuccessful. Schilling and Pearse[72] did find a greater incidence of ulceration in dogs after gastrectomy with pyloric exclusion than after a similar operation with excision of the antrum. This is contrary to the experience of most investigators who found essentially the same secretory activity in gastric pouches and a similar incidence of jejunal ulcers before and after excision of the pylorus. Food and other chemicals placed in isolated pyloric pouches did not stimulate activity in the fundic cells.[38, 46, 64, 79]

Alvarez[22a, 6] agrees with Priestley and Mann that, in the dog at least, the pylorus plays a minor part—if any—in the production of gastric acidity.

CLINICAL REVIEW

Clinical experience has been in almost direct contrast to the experimental findings. Further observations will be required to explain this apparently anomalous situation.

Several objections can be made to the previously recorded postoperative studies. (a) It is probable that in a number of cases rated as having a good

result with complete pylorectomy a fragment of stomach was actually left *in situ*. In 10 per cent of our cases microscopic study did not confirm the clinical impression of complete pylorectomy. Other more experienced surgeons have also found that very often it is impossible to delineate the pylorus *in vivo*.[26, 22b] (b) A similar question can be raised in cases of pyloric exclusion in which the entire mucosa was thought to have been excised. The difficulty and uncertainty of this procedure in the presence of an inflamed pylorus have been pointed out by several writers.[5, 26, 62, 73] (c) In the past there has been a tendency to attribute poor results to the presence of a

TABLE V.—*Recurrence Rates Reported for Cases Having Complete Pylorectomy or Mucosal Stripping.*

Authors	Year	Cases	Gastrojejunal Ulcers	Percentage of Recurrences
Brooks and Mecham[14]	1945	56	0	0
Graham[28]	1943	?	0	0
Kennedy and Reynolds[39]	1946	90	0	0
Miller[56]	1942	173	0	0
Steinberg[73]	1947	339	0	0
Wangensteen[83]	1947	500	2	0.4
Ransom[66]	1947	184 (gastric)	1	0.5
Colp *et al.*[19]	1948	100	1	1.0
St. John *et al.*[80]	1948	394	2 proved 3 suspect	1.2
Cline[18]	1943	78	1	1.3
Allen and Welch[5]	1946	195	3	1.5
Heuer[30]	1944	129	2	1.5
Garlock[26]	1949	168	3	1.8
Hinton[34]	1946	162	3	1.8
Marshall[53]	1944	318	6 (approx.)	1.9
Halloran Hospital	1949	85	2	2.3
Allen & Welch[3]	1942	142	4	2.7
Marshall & Devine[51]	1941	?	..	3.0
Gardner and Hart[24]	1948	89	4	4.5
Miller and Nicholson[57]	1946	108	5	4.6
Bartels & Dulin[11]	1947	60	4	6.7
Mage[50]	1942	600	41	6.8
Church and Hinton[15]	1942	97	9 suspect	9.3
Sanders[70]	1945	50	4	8.0
Reinhoff[67]	1945	260	12 proved 39 presumed	19.6

pyloric remnant when adequate proof was lacking. In one such series two-thirds of the stomach had been left in place as well as the antrum.[57] Another author presented a "proved" case of recurrent ulcer due to unexcised antral tissue, although the secondary operation included shortening of the afferent loop and resection of an additional 25 per cent of the stomach.[7] (d) Finsterer's recurrence rate has been cited as proof that the procedure is inadequate, although equally high recurrence rates have been reported for groups in which complete pylorectomy was done.[50, 67, 70]

Notwithstanding the objections listed above, a comparison of Tables V and VI shows a proportion of unfavorable results in the cases with antral exclusion which is probably beyond the range of coincidence or occasional

incomplete observation. In accepting these figures, we wonder with Schilling and Pearse[72] "why an excluded antrum increases the incidence of marginal ulcers (since) by exclusion the ordinary chemical and mechanical stimuli are removed."

TABLE VI.—*Recurrence Rates Reported for Cases with Antral Exclusion.*

Authors	Year	Cases	Gastrojejunal Ulcers	Percentage of Recurrences
Halloran Hospital	1949	16	0	0
Vale[78]	1942	6	0	0
Marshall[53]	1944	20	1 (?)	5.0
Finsterer[61]	....	...	..	4.8
Finsterer[60]	....	...	..	6.4
Lannin[46]	1945	not stated	..	8.5
Gardner and Hart[25]	1948	11	1*	9.1
Nissen[63]	1945	33	4*	12.1
Marshall and Devine[51]	1941	34	7	20.6
Steinberg[73]	1947	14	3	21.4
Ogilvie[43]	1938	22	6	27.3
Miller and Nicholson[37]	1946	not stated	4	....
Ransom[66]	1947	6†	3*	50.0
Allen and Welch[3]	1942	9	5	55.0
Garlock[26]	1949	1	1	100.0
Graham[9]	1947	4‡	4	100.0

* One case in each group failed to improve after antral excision.
† Gastric ulcer cases.
‡ Devine exclusion operation.

TABLE VII.—*Cases of Post-Gastrectomy Ulcer Treated by Excision of the Antrum Alone.*

Authors	Year	Results in Cases of Antrumectomy	
		Cured	Unimproved
McKittrick *et al.*[58]	1944	4	0
Allen[3]	1942	1	0
Marshall[54]	1946	3	0
Kiefer[40]	1942		
Schilling, Pearse[72]	1948	2	0
Wangensteen[82]	1945	2	1
[81]	1942		
Nissen[63]	1945	1	1
Bancroft[10]	1947	0	1
Forbes[23]	1943	0	1
Gardner and Hart[25]	1948	0	1
Ransom[66]	1947	0	1
Totals		13	6

The least equivocal proofs of the effect of the antral mucosa are those cases of gastrojejunal ulcer which have been cured by excision of the antrum alone. McKittrick and Moore[58] cited five such cases at Massachusetts General Hospital and two of Marshall. We have added six cases more recently reported. In addition six cases have been listed in which removal of the antrum did not prevent recurrent ulceration (Table VII).

COMPROMISE PROCEDURES

The majority of clinical evidence suggests that complete pylorectomy is a necessary though sometimes hazardous step in the performance of gastrectomy for ulcer. A number of compromise procedures have been devised in an effort to remove the antral mucosa without the employment of "meddlesome venturesomeness."[80]

Finsterer, in 1919, suggested excision of the mucosa before inversion of the antrum. He later abandoned excision of the mucosa; references to the "Finsterer operation" in the recent literature usually imply exclusion of the intact antrum. Bancroft introduced the original procedure into the American literature in 1932.[8] The operation is not easy, particularly in those cases for which it was specifically designed. A fairly high incidence of complications has been reported following it (Table VIII). Allen has said that the "antrum is a thin friable structure after its mucosa is eliminated and offers, under these

TABLE VIII.—*Reported Complications Following Antral Exclusion with Mucosal Stripping.*

Authors	Cases	Fistula	Abscess	Total
Wangensteen[83]	100 app.	0	0	0
Gardner and Hart[33]	12	0	0	0
Allen and Welch[3]	13	1	0	1
Bancroft[8]	14	0	1	1
Halloran Hospital	1	1	1	2
Wangensteen[80]	13	0	2	2
McKittrick *et al.*[58]	?	2	0	2
Steinberg[73]	?	7	0	7

circumstances, tissue that may not lend itself to suitable closure."[1] Wangensteen feels that it is fear of leakage which has prevented the universal adoption of mucosal excision. Two of his first 13 cases developed subphrenic abscesses. He later devised a method for obliteration of the antral remnant without inversion which, in his hands, has been successful.[83] In Garlock's[26] opinion "it is impossible to be sure that this coring out procedure thoroughly removes the mucosa from the pyloric ring." Steinberg[73] and Allen[5] have reported cases in which secondary operation proved the presence of residual antral mucosa in spite of apparently complete stripping at the original operation.

Because of the danger and uncertainty of mucosal stripping, McKittrick proposed a two-stage procedure, the first stage being a modified Devine transection with exclusion and the second stage a resection of a fairly large pyloro-antral remnant. He found considerably less edema and induration at the second operation. Nevertheless, in the original series of 37 patients there was a duodenal stump blow out in one case and complete transection of the common bile duct in another.[58] Lahey also advocates a two-stage operation in cases where there is serious doubt that the duodenum can safely be dissected. He favors either the operation described by McKittrick or a true Fin-

sterer resection with exclusion "with the understanding that the duodenum and pylorus will be removed at a later date when the ulcer has healed and the induration has disappeared."[44] He warns against this procedure in the presence of pyloric obstruction.

SUMMARY AND CONCLUSIONS

1. In a series of 101 cases at Halloran Hospital there were two frank duodenal stump blow-outs. One of them occurred in a case in which an inflamed antrum had been excluded and the mucosa stripped.

2. The preoperative acid and the height of the resection were the most important factors influencing the postoperative acid values. In general gastric ulcer cases had lower pre- and postoperative acids than duodenal ulcers.

3. Microscopic study proved that pylorectomy had been incomplete in 16 cases. No untoward clinical or laboratory effects have as yet been observed in this group.

4. Two patients had definite evidence of gastrojejunal ulceration. Three others had possible recurrences. Four of these had had resection of not more than 50 per cent of the stomach. All five had had complete resection of the pylorus and antrum.

5. A brief review of the literature revealed no convincing experimental evidence that the pylorus influences the secretion of hydrochloric acid by the fundus.

6. A review of the clinical literature indicates that complete pylorectomy is a requisite, though sometimes dangerous, part of an adequate gastrectomy for ulcer.

7. We cannot explain the apparent disparity between most clinical observations and the laboratory experiments, nor the failure of our own observations and those of a few others to coincide with the general experience.

8. Further observations and reports are desirable. They should include routine microscopic study of the distal specimen.

9. In those cases in which resection of an inflamed pylorus and antrum appears hazardous an alternative procedure should be chosen. The two-stage procedures described by McKittrick or Lahey offer the most certain, and sometimes the safest, means of completely excising the pylorus. If pyloric obstruction or peri-duodenal scarring is marked, exclusion of the antrum with excision of the mucosa may be preferable.

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NIPPLE DISCHARGE; ITS CLINICAL AND PATHOLOGIC SIGNIFICANCE*

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NIPPLE DISCHARGE is a relatively frequent symptom of a pathologic lesion in the non-lactating breast. Often it is the initial symptom which brings the patient to the physician for treatment. A difference of opinion exists as to the significance of the discharge, and this is particularly true if the discharge is sanguineous. Some authors[3, 4] consider the discharge as an innocuous symptom requiring only observation. Others consider it to be an indication of a lesion of a more serious nature requiring active surgical treatment, which may vary from local excision to mastectomy. Most observers place the greatest emphasis on the discharge rather than on the underlying pathologic lesion in the breast. A majority of reports[1, 5, 16-18, 21, 22] stress only the significance of a sanguineous discharge. While it is true that such a discharge is indicative of a lesion of a more serious nature, undue emphasis on the presence of blood tends to lead to a sense of false security if the discharge does not contain blood.

This discussion includes a study of all types of nipple discharge, and its purpose will be to stress that abnormal nipple discharge is a symptom of a pathologic change in the ductal and secretory systems of the breast, and only in a general way offers information as to etiology. It is to be emphasized that two persons will react differently to identical lesions. A papilloma may initiate a bloody discharge in one patient, a serous one in another, and no discharge in a third. Likewise, the same type of discharge may be associated with lesions considerably different in character. Each patient thus is an individual problem, and the correct clinical significance of the nipple discharge can only be reached after a careful consideration of the particular situation.

INCIDENCE

In a review of 2269 cases of mammary lesions treated in the Department of Surgery at the State University of Iowa Hospitals, in a 20-year period from 1929 to 1949, it was found that 219 patients, or 9.6 per cent had a presenting complaint of nipple discharge. This is slightly higher than the incidence reported elsewhere. For example, Hinckey[13] reported nipple discharge in 7.6 per cent of 898 patients seen. Of the 219 patients with nipple discharge, 15 patients, or 6.8 per cent, presented a bilateral nipple discharge.

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Generally speaking, abnormal nipple discharge can be regarded as a symptom occurring in the female. Only isolated examples occurring in the male have been reported.[12] Two of the patients in this series of cases, or .9 per cent, were males.

The age incidence was found to vary with the type of pathologic lesion producing the nipple discharge. For example, it will be shown later in the discussion that a clear serous discharge from the nipple occurring before the menopause may be on a secretory basis. When such a discharge occurs after the menopause, it must be considered in terms of epithelial overgrowth. The chronologic age relationship with the various pathologic lesions is shown in Table I. This relationship throws some light on the pathogenesis of the common lesions producing nipple discharge, which in turn influences the type of treatment to be instituted.

TABLE I.—*Average Age Incidence in Relation to Pathologic Lesion.*

Lesion	Age (average)
Chronic cystic mastitis	44 years
Intraductal papilloma	50 years
Intraductal carcinoma	58 years
Carcinoma	62 years

TYPES OF DISCHARGE

Pathologic lesions of the breast which involve the ductal and secretory systems of the breast produce a variety of abnormal discharges from the nipple. A study of the pathologic physiology of the breast enables one to understand better the formation of these discharges.

It should be remembered that the breast is not a static organ, but undergoes constant physiologic changes as the result of ovarian activity. These changes are manifested by a periodic cycle of hyperplasia and involution within the breast. In the premenstrual cycle, the epithelial cells of the ducts become larger and branch out into the periductal tissue. Associated with this expansion, there is retention of water in the periductal tissue, producing a myxomatous appearance. Lymphocytes appear in the periductal tissue. The breast being an apocrine gland, the epithelium develops vacuoles in the cytoplasm and albuminous secretion appears in the ducts. If pregnancy does not occur, involution takes place and the products of secretion, namely, desquamated epithelium, cellular debris, and protein precipitates are shed into the ductal system. Normally, these products undergo autolysis or are engulfed by the phagocytes present.

A disturbance of the above cycle, usually a result of relative ovarian hormone overbalance, may produce excessive hyperplasia or delayed involution, and with it the various manifestations of epithelial hyperplasia and abnormal involution within the breast commonly known as chronic cystic mastitis.

These changes may result in an accumulation and stagnation of exfoliative products within the ductal system.

Thus, the types of discharges in a general way fall into two main groups, namely, that in which the character of the discharge is determined by an apocrine-like exfoliation and stagnation within the ductal system, and that in which hyperplastic or neoplastic changes in the ductal system influence the character of the discharge. We can thus speak of the patient as having a stagnation discharge or a hyperplastic discharge. There will, of course, in some instances be transitions from one group into the other but in a general way this placing of patients in one of the two groups simplifies their evaluation.

Breast lesions which produce stagnation discharge are usually forms of chronic cystic mastitis without hyperplasia. The discharge is usually white,

TABLE II.—*Incidence of Types of Nipple Discharge.*

Type	Number of Cases	%
Serosanguineous or sanguineous	121	55.0%
Serous	47	21.0%
Yellow	24	11.0%
Green	7	3.3%
Whitish (milky)	13	6.0%
Brown	7	3.2%

yellow, green or brown and non-sanguineous in nature. In our series the green nipple discharge occurred most frequently in young women who had had several pregnancies. Microscopic examination of the discharge shows it to contain desquamated degenerated epithelial cells, colostrum cells, lymphocytes, mucus, and protein precipitate. In eight patients the discharge was cultured and *staphylococcus albus* was isolated in three cases and alpha hemolytic *staphylococcus aureus* in two. While these were probably skin contaminants, it may prove of value to culture more of the nipple discharges in an effort to determine how much of a rôle infection plays as an etiologic agent. In the patient with stagnation discharge, the discharge is frequently from more than one duct, may vary in color within the same breast, and is often bilateral.

In the hyperplastic group the underlying lesion is predominantly one of hyperplasia. This varies from heaping up of epithelium producing small papillary invaginations to definite intraductal adenoma, papilloma or carcinoma. The discharge is serosanguineous or sanguineous. Microscopic examinations show red blood cells to predominate. In addition, desquamated epithelial cells, tumor cells, lymphocytes, and protein precipitate may also be present. Some of the ducts contain a bronze pigment resembling hemoglobin. Chemically, tests are positive for blood.

One other breast lesion produces a characteristic discharge. The galactocele, a form of retention cyst, is observed in connection with obstruction

of the larger lacteal ducts. The discharge is milky and persists after the nursing of the child is completed. Microscopic examination revealed colostrum cells and desquamated epithelium. Chemical tests on the discharge from one patient for lactose and galactose were negative.

TABLE III.—*Lesions Producing All Types of Nipple Discharge in 219 Patients.*

Intraductal papilloma	66	89
Intraductal carcinoma	23	
Adenocarcinoma	69	
Chronic cystic mastitis	48	
Paget's disease	5	
Retention cyst of breast	2	
Galactocele	2	
Chronic inflammation	1	
Abscess of breast	2	
Carcinosarcoma	1	
	219	

ETIOLOGY OF NIPPLE DISCHARGE

Observations by others[5, 11, 13, 19] reveal that chronic cystic mastitis, intraductal papilloma, and carcinoma are responsible for approximately 75 per cent of the instances of nipple discharge. However, Paget's disease, sarcoma, inflammatory lesions, and fibroadenoma have all been reported as producing nipple discharge. Adair[2] noted that seven out of ten patients with plasma cell mastitis had a nipple discharge. Twenty-one per cent of patients treated for plasma cell mastitis at the Mayo Clinic[7] had nipple discharge as a presenting symptom.

TABLE IV.—*Relationship Between Type of Discharge and Pathologic Lesion.*

	Type of Lesion			
	Benign or Precancerous		Malignant	
Type of Discharge	Chronic Mastitis	Ductal Papilloma	Carcinoma	Paget's
Sanguineous	10*	50	59	2
Serous	5†	12	27	3
Yellow	17	2	5	..
Green	7	..	..	..
White (milky)	12	1	..	.
Brown	4	1	2	..

* Histologic lesion in 10 patients was chronic mastitis with hyperplasia.
† Histologic lesion in 3 patients was chronic mastitis with hyperplasia.

A variety of lesions were responsible for nipple discharge in 219 patients treated by the Department of Surgery at this hospital. These are shown in Table III. In this series of cases intraductual papilloma, chronic cystic mastitis, and carcinoma are also the main etiologic lesions, comprising 90 per cent of those which produced nipple discharge.

Table IV shows the relationship between the type of discharge and the underlying breast lesion producing it. This table illustrates very well that no one type of discharge is specific for any particular lesion. The same lesion may produce a variety of discharges, and the same type of discharge may occur with lesions considerably different in character.

Of the 115 patients with sanguineous discharge, 61, or 53 per cent, were found on histologic examination to have a malignant lesion. The remaining 54 patients, or 47 per cent, had a benign or precancerous lesion. What is most significant is that in all the patients with a sanguineous discharge, histologic examination of the lesions showed evidence of epithelial overgrowth, namely, hyperplasia. On this basis it is important that it be determined in the initial examination if the nipple discharge contains blood. Its presence is an indication of hyperplasia and may be a warning sign of a precancerous or malignant lesion in the underlying breast. It is important to keep the relationship of sanguineous discharge and hyperplasia in mind when treatment of the patient with nipple discharge is considered.

This table also points out the mistake of undue emphasis on the exclusiveness of the sanguineous nature of the discharge, for 64 per cent of the patients with serous discharge also had malignant lesions. In this respect, it should be pointed out that these patients develop their nipple discharge sometime after the menopause. This once again emphasizes the importance of age in evaluating the nature of the lesion producing nipple discharge. A similar discharge occurring before the menopause would normally be associated with those lesions which produce a stagnation type of discharge. It should also be noted that in some of these patients the discharge occurred with obvious cancer of the breast, and was of no great significance from the diagnostic standpoint. The discharge in such patient was thus an associated finding.

PATHOLOGY

A correct clinical interpretation of nipple discharge is dependent upon one's knowledge of the nature of the underlying lesions producing it. As was previously stated, abnormal nipple discharge is indicative of a pathologic change within the ductal and secretory systems of the breast. The amount, color, odor, and consistency depend not only on the pathologic lesion, but on the degree to which the secretion is altered before reaching the surface of the nipple.

An eroded vessel wall bleeds, whether at the tip of a benign papilloma or adjacent to or within a carcinoma. If the bleeding lesion is situated near the orifice of the duct, the escaping fluid is frankly hemorrhagic. If deep within the matrix, the fluid assumes a serosangineous or a brownish color due to the degeneration of retained blood and its admixture with other secretions from the duct. The character of the discharge may also be altered by infection or necrosis.

A proper understanding of the intraductal papilloma more than any other lesion enables one better to understand nipple discharge. It is the lesion which creates the greatest problem as to proper diagnosis and treatment. The papilloma histologically is a complex papillary mass projecting into the lumen of a duct. The papillary mass is made up of branching stromal bands and is usually attached to the wall of the duct at one point. These stromal bands are coated over with epithelium similar to that of the wall of the duct which is usually cuboidal. Since the stroma is vascular, oozing may occur from its surface producing the blood-stained or frankly bloody discharge.

It is important to recognize that the papilloma histologically may vary from mere heaping up of epithelium to a complex papillary pattern producing a mass one centimeter or more in diameter. In the earlier stages it is too small to be palpated or to be visualized by transillumination but may still produce nipple discharge, usually of the hyperplastic type. The patient with bleeding from the nipple, and no palpable mass, usually has this type of histologic lesion. There can be transitions from one type into another, and several of the types can be found within one breast (see Fig. 3). This transition helps to explain the pathogenesis of lesions which produce nipple discharge, and in turn fits in with the chronologic age relationship that the common lesions which produce nipple discharge show.

Multiplicity of intraductile papillomas is frequent. The exact frequency is unknown as such statistics would require the complete excision of all breast tissue with many sections of tissue studied. Even with incomplete studies 20 per cent of the papillomas in patients treated in this hospital were multiple in character. This has also been the experience of others[11, 22] who have studied patients with this type lesion. Where such a high percentage of papillomas occur as multiple lesions it would seem that, unless a whole section of breast be examined microscopically, it would not be possible to state whether a particular papilloma is the only one present.

Four patients in this series of cases treated by local excision of a papilloma, which histologically showed evidence of hyperplasia, had recurrences. Two patients with chronic mastitis with hyperplasia treated by local excision, also had recurrences. In each case the recurrence was a malignant change. This lends support to those who advocate simple mastectomy as the treatment of choice for the patient with intraductal papilloma. The high incidence of multiple occurrence of papilloma should certainly be kept in mind when treatment of this lesion is considered. It seems well established that papilloma of the breast may form part of a process in which carcinoma may develop. Considerable time may elapse, however, before this occurs, which could easily serve to explain why the uncomplicated papilloma is most often seen in young and middle-aged women, and papillary duct carcinoma occurs in an older age group. Both lesions occur in identical locations.

In cancer of the breast, nipple discharge results from invasion of the ductal system by the neoplasm. The character of the discharge is influenced

FIG. 1

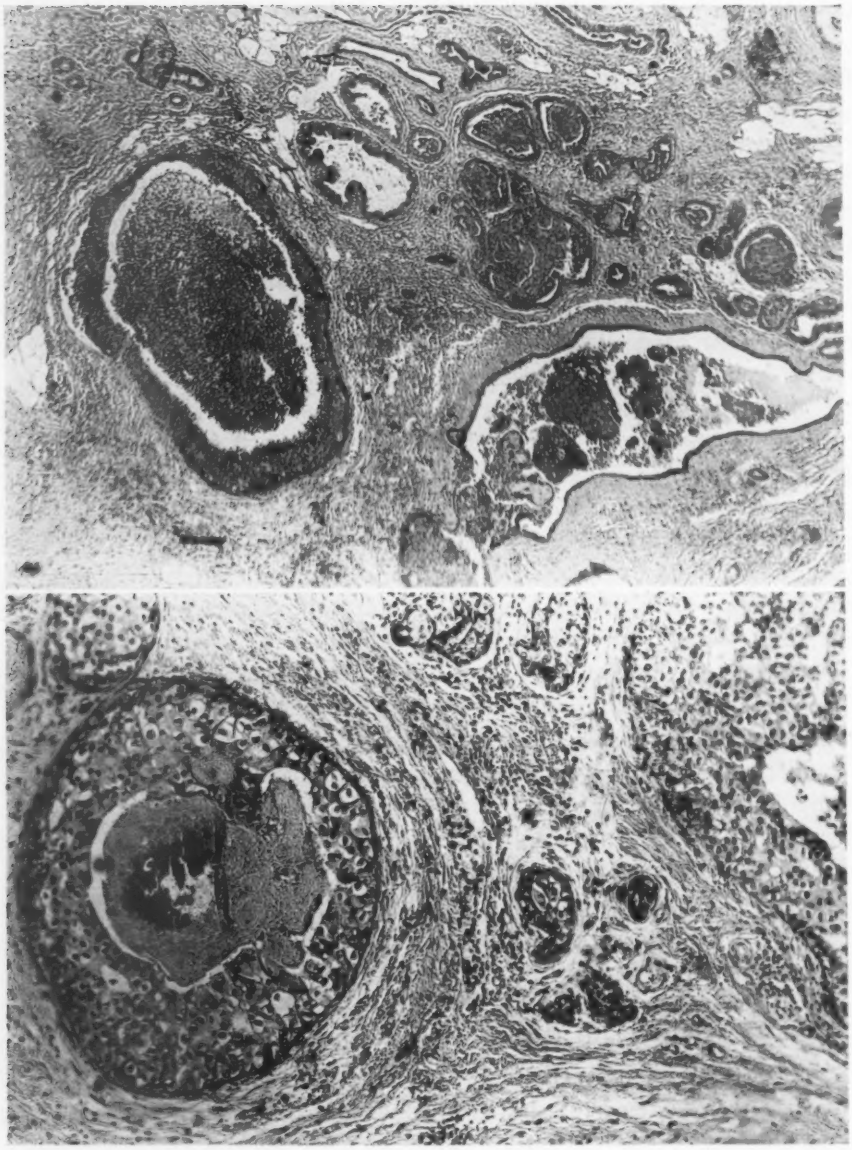

FIG. 2

FIG. 1.—Chronic mastitis with benign hyperplasia. Note red blood cells in lumen of duct; also multiple intraductal papillomas.

FIG. 2.—Chronic mastitis with precancerous hyperplasia. Patient clinically showed a sanguineous nipple discharge without a palpable mass present.

by such factors as infections, necrosis, and erosion of blood vessels. Nipple discharge may be the initial and sometimes the only symptom of a malignant breast lesion. Figures 1 through 4 show photomicrographs of breast lesions which produced nipple discharge. Figure 3 is of exceptional interest in that it shows hyperplasia, intraductal papilloma, and carcinoma within the same breast.

DIAGNOSIS

The patient with abnormal nipple discharge requires careful study to determine the nature of the underlying lesion. Such study includes a careful history, physical examination, microscropic examination of the stained smears of the discharge, and transillumination of the breast. In the majority of instances one may arrive at the correct clinical interpretation of the discharge and institute proper treatment.

The symptom of nipple discharge is frequently the initial symptom which moves the patient to consult a physician. This is especially true if the discharge is bloody in type. Examination of the breasts in these patients may or may not reveal the presence of a tumor. The patient without a palpable mass has produced the greatest difference of opinion among physicians as to proper treatment. In others, one or more masses may be present or the breast may present a diffuse nodularity. Again, obvious signs of carcinoma may be present. Except in those patients with obvious carcinoma, the routine in arriving at a diagnosis is essentially the same.

Some patients present themselves stating that they have a bloody discharge from the nipple when actually it is a yellow or brown color and nonsanguineous in type. The only sure way one can determine if he is dealing with a sanguineous discharge is to find red blood cells on microscopic examination. A positive chemical test lends further confirmation. It is important to establish the sanguineous nature of the discharge, for in our experience its presence indicates there is about 50 per cent chance that the underlying pathologic lesion is malignant. In those patients having a sanguineous discharge without a palpable mass, 36 per cent on histologic examination revealed a lesion showing malignant or precancerous change. Also of importance is the fact that where cancer is absent, it is an indication that some degree of hyperplasia is present. This in turn influences the type of treatment to be instituted.

Microscopic examination will on occasion reveal malignant epithelial cells and give a clue to the underlying lesion present, such as an intraductal carcinoma. Jackson and Severance[15] studied in detail nipple discharge from 974 breasts. On three occasions a carcinoma was found where diagnosis had been made preoperatively from a study of the nipple secretion. The authors stress that cytologic study of breast secretions is an aid in the diagnosis but by no means should supplant biopsy or other diagnostic means. Our experience with this procedure is less extensive and to date is also less enthusiastic.

FIG. 3

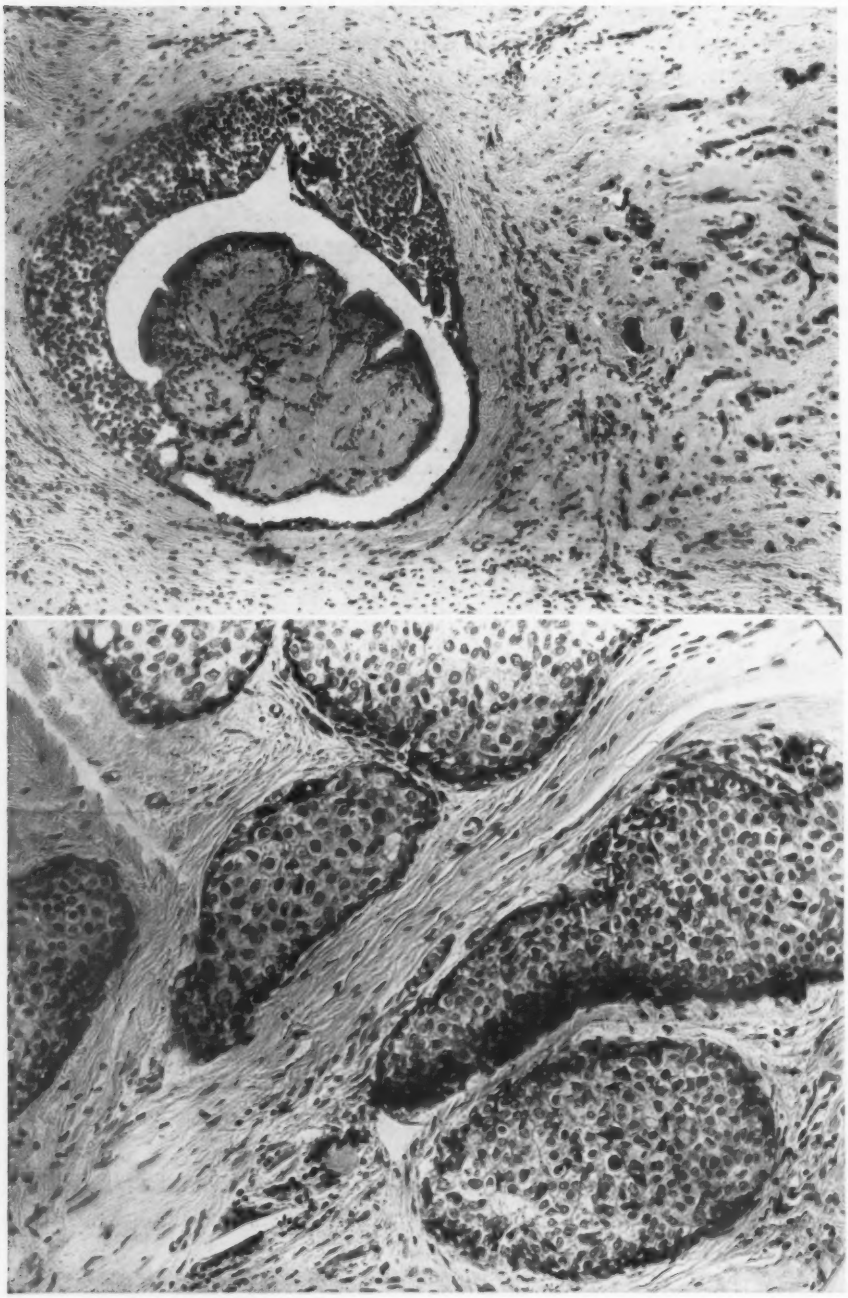

FIG. 4

FIG. 3.—Photomicrograph showing epithelial hyperplasia, intraductal papilloma, and carcinoma within same breast.

FIG. 4—Photomicrograph showing intraductal carcinoma. Clinically, patient had a sanguineous nipple discharge.

Transillumination of the breast has been reported by Cheatle and Cutler[6] as a valuable diagnostic aid. The method is based upon the varying translucence and opacity of different tissues. Fat is highly translucent; consequently, the breast transilluminates very satisfactorily. Blood has an intense opacity. Thus a papilloma with bleeding shows an opaque shadow. Such a procedure is supposed to be of aid in localizing a papilloma too small to be palpable or in revealing the presence of more than one papilloma within the breast. A solid tumor on transillumination is less intense than a papilloma and not as sharply outlined. It has been the experience at this hospital, however, that transillumination of the breast has been most disappointing as a diagnostic aid and has to a large extent failed to live up to the above-mentioned possibilities.

In the patient with nipple discharge the location of the involved duct can often be determined by careful palpation. Gentle digital pressure of the terminal ducts should be applied in a systematic fashion beginning at the periphery of the breast and examining the radii of the breast in a clockwise fashion. As has been mentioned previously, location of the mass and the patient's age may prove to be further aids in diagnosis.

TREATMENT

Inasmuch as a difference of opinion exists among physicians as to the significance of nipple discharge, there also exists a difference of opinion as to the preferred treatment for a patient with nipple discharge. The greatest controversy centers around treatment of the patient who has bleeding from the nipple without a palpable mass in the breast. Bloodgood[3] has expressed the opinion that "bleeding is only a symptom, and without a palpable tumor does not demand treatment." He further expressed the opinion that such a patient runs no greater risk of developing cancer. His opinion was based on cases with nipple discharge that had received no treatment, and which he followed; who either had a spontaneous cessation of the discharge or no other signs or complications developed. Campbell[5] and Slaughter and Peterson[19] in recent articles have stressed the desirability of more conservative treatment in the patient with nipple discharge.

In disagreement with the conservative treatment are those authors[13, 14, 20-22] who regard the whole breast with suspicion and recommend more active surgical treatment. Their recommendations are based on experience which has included patients in whom nipple discharge preceded a malignant lesion; patients who had nipple discharge but no mass in the breast who were observed for a period of time and then developed carcinoma; and patients who showed a benign papilloma in one area of the breast and malignant in another. Warren[23] studied the development of carcinoma in 1206 women whose breasts had been operated upon for benign lesions and made a comparison with the incidence of breast carcinoma in females of general population in the same age group. He concluded that women with breast abnor-

malities, in particular those with papillary cystadenoma, show a definite predisposition to the development of cancer. This was true despite the fact that the most abnormal or even all of the apparent abnormal tissue had been supposedly removed by surgical intervention.

In general, our findings are more in accordance with those authors who advocate more active surgical treatment rather than those who advise periodic observation of the patient with nipple discharge. Of the 219 patients treated at this hospital, 36 or 17 per cent did not have a palpable mass when examined. All but one of these patients were operated upon, and the type of lesions found on histologic examination (see Table V) are of definite interest, in that 30 of the lesions showed definite evidence of hyperplasia. Five patients had malignant lesions. Six patients had lesions showing definite precancerous change.

Twenty-one of our patients gave a history of having nipple discharge from three months to nine years prior to the presence of a mass in the breast.

TABLE V.—*Histologic Lesions in 35 Patients Operated Upon with Nipple Discharge and No Palpable Mass.*

Type of Discharge	Number of Cases		Type of Lesion
Hyperplastic			
Serosanguineous or sanguineous	30	5	Chronic mastitis with benign hyperplasia
		3	Chronic mastitis with precancerous hyperplasia
		3	Intraductal papilloma with precancerous hyperplasia
		14	Intraductal papilloma
		4	Intraductal carcinoma
		1	Paget's
Stagnation			
Yellow	1		Chronic mastitis
White	1		Infected retention type
Green	2		Chronic mastitis
Milky	1		Galactorrhea

Two of these patients when examined were found to have carcinoma with osseous metastases. The remaining 19 patients had operable lesions, and the histologic examination revealed the following lesions: 12 adenocarcinomas, six intraductal in origin; one Paget's disease with underlying intraductal carcinoma; four intraductal papillomas, and two chronic cystic mastitis with hyperplasia, one precancerous in type. Another patient with a history of bleeding nipple was observed and not treated over a ten-year period. Here, also, carcinoma of the breast with bony metatases developed. This lends additional support to those cases reported in the literature of bleeding nipple preceding a carcinoma. These findings suggest that serious results could have occurred in a number of the patients who had a bloody discharge but no mass if they had been observed and not treated until a definite mass appeared. In the carcinomas, the stage of operability could well have been passed, and some of the precancerous lesions could possibly have undergone malignant change.

It seems certain that if the 12 patients who had a discharge prior to the appearance of the cancer had been treated surgically before the mass was detected, the end result would have been definitely better, for a number of these patients had axillary metastases at the time of operation.

TABLE VI.—*Results of Treatment of Patients with Nipple Discharge.*
TABLE VI.—(*Continued*).

Lesion	Treatment	Number of Cases	Result
Bleeding nipple no mass	No treatment	1	After 10 years developed carcinoma with bony metastases.
Chronic cystic mastitis with hyperplasia	Local excision	5	(1) Well after 18 months. (2) Well 2 years. (3) Recurrence 5 years later, intraductal carcinoma. Treated by local excision. Recurred 1 year later and simple mastectomy done. (4) Developed bilateral carcinoma 6 years later. (5) Developed carcinoma of breast 12 years later.
Chronic cystic mastitis	Simple mastectomy	11	(1, 2, 3) Well 6 months. (4) Well 1 year. (5) Well 18 months. (6) Well 2 years. (7) Well 4 years. (8, 9) Well 6 years. (10) Well 7 years. (11) Well 8 years.
Intraductal papilloma	Local excision	7	(1) Recurrence 3 years later with carcinomatous change. (2) Well 8½ years. (3) Local excision. New lesion 4 years later treated by local excision; 13 years later carcinoma of the breast—radical mastectomy. (4) Recurrence 5 years, later intraductal carcinoma. (5) New lesion 3 years later—local excision. Now well 9 years. (6, 7) Well 13 years.
Intraductal papilloma	Simple mastectomy	23	(1) Well 3 months (2, 3) Well 6 months. (4) Well 1 year. (5, 6, 7) Well 2 years. (8, 9) Well 3 years. (10, 11, 12) Well 5 years. (13, 14) Well 6 years. (15, 16, 17, 18) Well 7 years. (19) Well 7 years. (20, 21, 22) Well 8 years. (23) Well 9 years.
Intraductal papilloma		3	(1) Died 18 months. (2) Well 5½ years. (3) Well 11 years.

Of the 104 patients with a diagnosis of chronic cystic mastitis with hyperplasia, intraductal papilloma, and intraductal carcinoma, it was possible to obtain a follow-up on 61 cases. No attempt was made to obtain a follow-up on patients with obvious carcinoma where nipple discharge was only an incidental finding. Also, no attempt was made in the cases of chronic cystic

mastitis which had a yellowish or greenish discharge and histologic examination revealed no evidence of hyperplasia. The results of follow-up are shown in Table VI.

Of significance is the fact that the patients who got into difficulty were those treated by local excision of the mass. In one instance, a precancerous lesion recurred five years later as a definite intraductal carcinoma. This was treated by a second local excision. There was recurrence one year later and simple mastectomy was then performed. It would appear that simple mastectomy at the original operation would have avoided two additional operations for this patient. Five other patients with precancerous change treated by local excision of the lesion also developed carcinoma subsequently. One other patient treated by local excision developed a benign recurrence with no difficulty after a second local excision. Simple mastectomy proved to be adequate treatment in all cases except one in which the patient with intraductal carcinoma died with lung metastases after eight years.

In view of the experience at this hospital, we recommend the following treatment for patients with nipple discharge:

1. *Discharge from the nipple and no palpable tumor.* The evidence presented that a hyperplastic discharge from the nipple can precede a malignant breast lesion, and that carcinoma has developed in patients with such a discharge who were observed over a period of time, and that a patient with sanguineous or serous discharge has approximately a 50 per cent chance of having or developing cancer in that breast, makes simple mastectomy the treatment of choice. The exception to this would be the patient with a stagnation type of discharge in which the history and clinical symptoms, in addition, makes a diagnosis of galactorrhea, or chronic cystic mastitis, obvious.

2. *Discharge and palpable tumor.* In the breast where a single palpable tumor is present local excision is recommended. If histologic examination reveals a precancerous lesion simple mastectomy should be performed; if malignant, radical mastectomy.

3. *Discharge and diffuse nodularity.* If the discharge is of the stagnation type and the clinical findings are those of chronic cystic mastitis, the treatment is dependent upon one's attitude toward the relationship of non-hyperplastic cystic mastitis and cancer. If a papilloma can be demonstrated, in view of what has been stated about the proportion of papillomas being multiple in occurrence, and the possibility of a benign papilloma occurring in one area of the breast, and a malignant lesion in another, simple mastectomy is recommended.

4. *Discharge with multiple masses.* In this type of patient the whole breast should also be viewed with suspicion and simple mastectomy performed.

SUMMARY

1. Abnormal nipple discharge is a relatively frequent symptom of a lesion of the non-lactating breast.

2. There is no specific nipple discharge for any of the varied breast lesions which produce discharge. The same lesion may produce a variety of discharges, and the same type of discharge may occur with lesions different in character.

3. In a general way, nipple discharge may be considered as falling into two main groups, namely, stagnation discharge and hyperplastic discharge. A knowledge of the possible pathologic lesions is essential for correct interpretation of the discharge.

4. Sanguineous nipple discharge with or without a mass is indicative of hyperplasia within the breast.

5. Chronic cystic mastitis, intraductal papilloma, and carcinoma account for the majority of lesions producing nipple discharge.

6. Treatment should consist of excision of any single palpable tumor with more radical surgery performed if indicated by histologic examination. Simple mastectomy is the treatment of choice in the patient with a bloody discharge from the nipple without a palpable mass.

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AORTIC VASCULAR RINGS ENCOUNTERED IN THE SURGICAL TREATMENT OF CONGENITAL PULMONIC STENOSIS*

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RECENT ADVANCES in the surgical treatment of patients with congenital heart disease have been responsible for the visualization of many anomalies of the great vessels previously not reported or seen only rarely in the dissecting room.[1] Gross and Ware[2] described the surgical treatment of anomalies of the aortic arch which may cause tracheal or esophageal obstruction. In such instances all or part of a vascular ring encircles the trachea and esophagus. It is the purpose of this report to describe the variations of such a vascular ring encountered in the surgical exploration of 841 patients for suspected congenital pulmonic stenosis.

A large and unexpected number of anatomic variations have been encountered in the treatment of congenital pulmonic stenosis, an example of the known fact that congenital defects are apt to be multiple. Variations of an aortic vascular ring have been encountered in 40 instances and other cases have probably escaped our notice. The observed cases have taken one of three forms: (1) A subclavian artery which arises from the medial side of the aortic arch as the last branch and passes behind the esophagus to the arm contralateral to the side of the aortic arch; (2) a retro-esophageal innominate artery; and (3) the persistence of both right and left embryologic fourth aortic arches.

EMBRYOLOGY†

During the first three weeks of embryonic life six aortic arches join the ventral aortic sac and the dorsal paired aortas around the interposed pharynx. The aorta caudal to the branchial arches becomes fused as an unpaired dorsal aorta. Figure 1-A shows diagrammatically the approximate configuration of the aortic arches in the 12 mm. embryo. The embryonic subclavian artery is the seventh cervical segmental artery which supplies the limb bud. As the heart and aortic sac move caudad and the cranial portion of the embryo elongates, the subclavian artery moves craniad on the aorta. Normally the caudalmost segment of the right fourth arch becomes obliterated as the left fourth arch takes over the majority of the cardiac output. Most of the right fourth arch becomes the first portion of the subclavian artery. The right third arch persists as the proximal part of the carotid artery. It ultimately arises from the fourth arch, which persists as the innominate and subclavian arteries. The left fourth arch persists as the definitive aorta, and the carotid and sub-

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† Based on Congdon.[3]

clavian arteries arise independently. In rare instances the left subclavian artery may ascend to the third arch, and a left innominate artery results.

If the left instead of the right arch becomes obliterated, a right aortic arch results, an anomaly which has been present in about 20 per cent of patients with congenital pulmonic stenosis. If the proximal rather than the distal part of the right fourth arch becomes obliterated, the right subclavian artery arises from the unpaired aorta and courses behind the esophagus to reach the right arm (Fig. 1B). In rare instances the vessel may go between the trachea and esophagus or anterior to the trachea.[4] A short segment of the distal end of

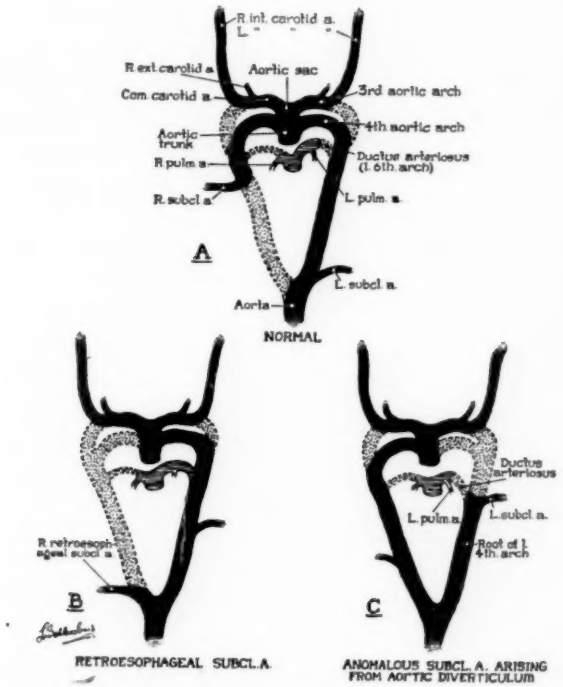

FIG. 1.—Development of aortic arch and anomalies. Diagram of aortic arches in 12 mm. embryo. Stippled segments disappear. See text.

the right fourth arch may persist as an aortic diverticulum from which the subclavian arises. If the right aortic arch persists and the left arch becomes obliterated, the mirror image of the above described arrangement may occur.

The ductus arteriosus usually connects the main or the left pulmonary artery with the left aortic arch. When the proximal left fourth arch becomes obliterated, the ductus arteriosus is connected with the distalmost portion of the left dorsal aorta along with the subclavian artery (Fig. 1C). If the arch becomes obliterated distal to the ductus arteriosus, this relationship is lost.

When both aortic arches persist, a complete aortic ring is formed about the trachea and esophagus. In such instances the brachiocephalic vessels usually

arise from the arches independently, although double aortic arches with an innominate artery have been described.[5, 6]

REPORT OF CASES

A retro-esophageal subclavian artery has been encountered in 36 patients in our series. The anomaly was equally distributed between the right and left subclavian arteries. In no case was there a history of tracheal or esophageal obstruction, although attacks of cyanosis and dyspnea attributed to the congenital cardiac deformity may have been partially caused by the anomalous vessel.

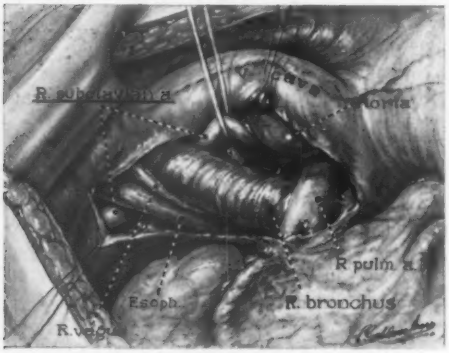

FIG. 2

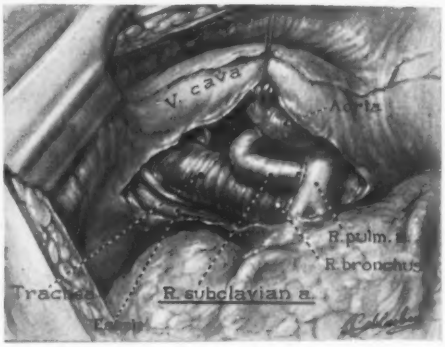

FIG. 3

FIG. 2.—Anomalous right subclavian arising from descending aorta and entering right chest behind esophagus. Reproduced by permission from Surgery, Gynecology and Obstetrics, **87**: 100, 1948.

FIG. 3.—The artery shown in Figure 2 was transposed to a position anterior to the right bronchus and esophagus before anastomosis to the pulmonary artery. Reproduced by permission from Surgery, Gynecology and Obstetrics, **87**: 100, 1948.

The usual appearance of a retro-esophageal right subclavian artery is shown in Figure 2. The vessel arises from the medial side of the left descending aorta and enters the right chest behind the esophagus. In no case in this series has such an anomalous vessel passed between the trachea and esophagus or anterior to the trachea. An anastomosis may be performed without altering the position of the subclavian artery or it may be brought anterior to the bronchus and esophagus as shown in Figure 3. A retro-esophageal subclavian artery is well adapted for the creation of an artificial ductus arteriosus.

An unusual type of anomalous left subclavian artery is shown in Figure 4. There is an aortic diverticulum from which the subclavian artery and obliterated ductus arteriosus arise. The anastomosis of the subclavian artery to the pulmonary artery is shown in Figure 5.

In one instance the innominate artery arose from the aorta and passed to the left behind the esophagus.[1] Its subclavian branch was suitable for an anastomosis.

The presence and course of the ductus arteriosus has been variable. In only several instances has it been connected to a retro-esophageal vessel. When present, the recurrent or inferior laryngeal nerve loops around it. If there is no ductus arteriosus, the inferior laryngeal nerve may enter the larynx directly,

FIG. 4

FIG. 5

FIG. 4.—Unusual retro-esophageal left subclavian artery arising from an aortic diverticulum with obliterated ductus arteriosus.

FIG. 5.—Anastomosis of the subclavian artery shown in Figure 4 to the left pulmonary artery.

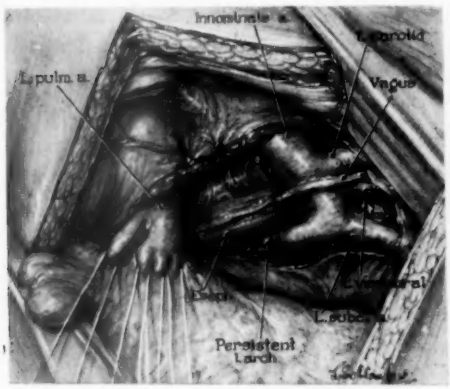

FIG. 6

FIG. 7

FIG. 6.—Left side of double aortic arch. The size of the vessel diminishes beyond the origin of the left subclavian artery.

FIG. 7.—Anastomosis performed using divided end of left aortic arch shown in Figure 6 and the left pulmonary artery.

loop around the vertebral artery, or loop around the inferior thyroid artery.[7] In such instances, of course, the nerve is not seen during thoracotomy.

There were three patients who had a complete aortic ring. The diagnosis in the first patient was mentioned at operation but not appreciated until she died of a ball embolus 14 days later. The patient had a marked scoliosis, and before operation there was considerable doubt about the side of the aortic arch. At

operation on the right side, deviation of the descending right aortic arch to the left was attributed to the scoliosis. This patient has been reported previously.[1]

The second patient was an eight year old boy with history and physical findings typical of the tetralogy of Fallot. Fluoroscopy with barium swallow demonstrated a right aortic arch with a retro-esophageal vessel. It was thought that this vessel might be a left descending aorta or more probably a small left subclavian artery. At operation (Figs. 6 and 7) the innominate artery was as usual the first branch of the right aortic arch. It gave rise to the left carotid and subclavian arteries and another vessel which was probably the left vertebral. In addition, there was a vessel which descended behind the esophagus. Upon occlusion of this vessel it was learned that a pulsating flow entered the vessel from either end and there was no diminution of pulses in the legs. The junction of this vessel with the aorta could be felt but not seen. It was thought that the artery represented a persistent left aortic arch. It was divided at its caudal end and the proximal end anastomosed to the left pulmonary artery. The patient was improved and his subsequent course has been satisfactory.

The other patient was a six-year-old boy. Throughout his life he had experienced frequent upper respiratory infections. He often had a loud barking cough. There were frequent fainting spells which had decreased with increasing age but occurred about twice a month at the time he was first seen. He was occasionally cyanotic at rest and definitely but not strikingly so on exertion. The physical findings were those of the tetralogy of Fallot with moderate incapacity. Oximeter studies showed a fall of his arterial oxygen saturation from 88 per cent to 84 per cent on exercise. Cardiac catheterization and angiocardiography were compatible with the diagnosis of tetralogy of Fallot but no information was obtained about the aortic arch. Roentgenography with barium swallow showed bilateral esophageal indentation in the anterior-posterior projection and indentation from behind in both the right and left anterior oblique positions. It was thought that the main aortic arch was on the left side, but that a double aortic arch had been responsible for the fainting spells in the absence of marked cyanosis and anoxemia. At operation on the left side a double aortic arch was found. The anterior and smaller arch was the left from which the subclavian artery arose. The origin of the left carotid artery was not seen, but presumably it arose from the left arch. Pulsations were noted at both ends when the arch was occluded. An obliterated ductus arteriosus joined the left aortic arch and the left pulmonary artery. Division of this structure partially relieved tracheal constriction. The arch was divided and the proximal end was closed proximal to the origin of the left subclavian artery. It was thought the subclavian artery could be used in an anastomosis but on attempting to close the distal end of the arch the subclavian artery was partially constricted. Consequently the subclavian artery was sacrificed and the distal end of the arch anastomosed to the left pulmonary artery without undue tension. Division of the left aortic arch seemed to relieve all respiratory distress. After operation he showed evidence of respiratory obstruction, and it was feared that manipulation and traction on the left recurrent nerve was

responsible. A temporary tracheotomy was done, and his subsequent course has been without complications. The cyanosis has disappeared, and the difficulty in breathing has been relieved. The findings in this case are illustrated in Figures 8 and 9.

DISCUSSION

The diagnosis of a retro-esophageal artery can usually be made by roentgenography with barium swallow.[8] The anomalous vessel causes an abnormal indentation in the posterior wall of the esophagus seen in the customary oblique and lateral projections. To determine the type of vessel is not always so easy. The size and level of the indentation are important. The most

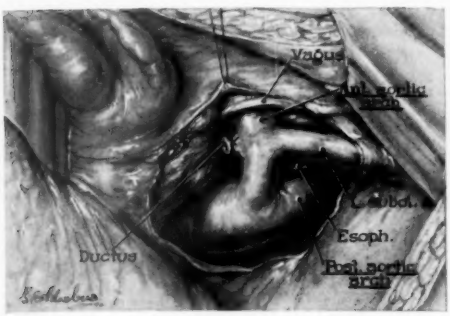

FIG. 8

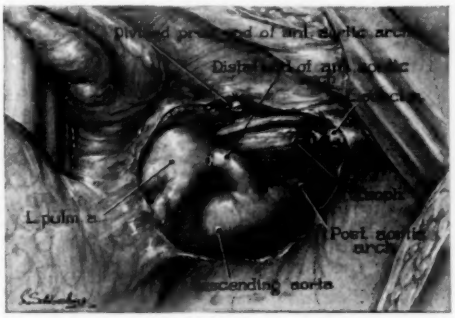

FIG. 9

FIG. 8.—Left side of double aortic arch causing tracheal obstruction. Ductus arteriosus divided.

FIG. 9.—Anastomosis performed between distal end of left aortic arch as shown in Figure 8 and left pulmonary artery.

common retro-esophageal vessel has been an anomalous subclavian artery. A retro-esophageal subclavian artery arising from an aortic diverticulum may give the same roentgenographic picture as a double aortic arch since the size and location of the aortic diverticulum may be similar to the distal end of a persistent double aorta. Large collateral arteries from the aorta to the lungs in the presence of pulmonic atresia are a fairly common cause of indentation of the esophagus. Angiocardiography may demonstrate the anomalous vessel but this diagnostic aid has been of little help in this series. It has been of great value in demonstrating the side of the aortic arch in several unusual patients.

It is important to recognize the presence of a retro-esophageal subclavian artery at the time of operation for pulmonic stenosis and appreciate that it may be used in creating an artificial ductus arteriosus. In three instances early in this series of patients the anomaly was not recognized and the carotid artery with its greater operative risk was used.

In cases of double aortic arch the operative approach probably should be made on the side of the smaller arch if the two are of unequal size. If this is

done, the subclavian branch may be used for the anastomosis or the arch may be divided and one of the ends employed for this purpose. If the ring is responsible for tracheal or esophageal obstruction, it is, of course, important that the ring be divided and the obstruction relieved.

SUMMARY

Variations of a vascular ring due to anomalies of the aortic arch encountered in the surgical treatment of patients with congenital pulmonic stenosis are described. A brief description of the embryologic development of these anomalies is given. Suggestions are made in regard to the use of a retroesophageal subclavian artery for the creation of an artificial ductus arteriosus.

Three cases of double aortic arch are described. In one of these patients the diagnosis was suspected at operation but not proved until later. In the other two patients the smaller arch was divided and one end was anastomosed to a pulmonary artery.

Marked respiratory difficulty as a result of tracheal constriction was present in only one patient in this group and was corrected by division of the smaller of the two arches.

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THORACIC DIVERTICULA WHICH ORIGINATE FROM THE INTESTINE*

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THERE HAVE COME to our attention three cases of unusual nature, in each of which a hollow structure within the chest proved to be an elongated diverticulum which came through the diaphragm and which was found to have an origin from the small intestine. In spite of a wide experience with congenital malformations, we have not previously encountered such an anomaly, nor have we found descriptions of it in the literature. We are therefore prompted to publish a note regarding the symptomatology and the surgical treatment of these patients.

In each of these cases there arose from the duodenum or from the jejunum a side-arm which left the intestine at a right-angle and coursed upward to the right crus of the diaphragm, which it pierced. Parts of such a side-arm had the general diameter, thickness and appearance of a loop of the patient's intestine, but in some places it ballooned out into quite a voluminous structure. As it ran superiorly to reach the diaphragm, the diverticulum always had a deep-lying position behind the stomach or behind the gastrohepatic ligament. In two cases, a diverticulum in the jejunal mesentery was close to branches of the superior mesenteric vessels, and then passed anterior to the pancreas. In the third case, a diverticulum (from the first part of the duodenum) ran upward and slightly backward to reach an area behind the vena cava.

All three of these diverticula penetrated the right side of the diaphragm and thereafter continued upward in an extra-pleural location in the posterior part of the chest. While they projected into and encroached upon the pleural cavities, they were always covered by parietal pleura. Above the diaphragm, the diverticula assumed multiple forms; either they remained as a narrow, tubelike structure or else they widened out into a more capacious and rounded mass. The intra-thoracic extent of these anomalies was a remarkable feature, for in all instances they rose to the very apex of the chest. In our first case, the diverticulum was long enough to be coiled upon itself, lying just above the diaphragm and to the right of the heart. The structure then coursed upward, took a sharp angulation to the left, ran behind the esophagus and the aorta, turned upward and proceeded to the left apex of the thorax. In our next case, the diverticulum was about the diameter of intestine as it came up through the diaphragm, but then quickly expanded into a more globular form,

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the central part of which was compressed by the azygos vein, giving it a dumbbell shape. This hollow mass extended to the right thoracic apex and had a volume which was more than half that of the right pleural cavity. In our third patient, the diverticulum in the lower third of the chest had the diameter of intestine, its middle third had a bulbous enlargement, while the upper third diminished again to a tube about twice the size of intestine.

The blood supply of these diverticula, while coming partly from the mesentery of the jejunum or from the duodenum, did not all arise from such sources. It was obvious that numerous arteries and veins joined them from the diaphragm and from many parts of the mediastinum.

The sizes of the diaphragmatic defects were of some interest. In our first case, the opening was large enough to permit herniation of several loops of ileum up into the chest (alongside of the diverticulum). In the other two cases, the diverticula pierced the diaphragm through a small opening, the edges of which were firmly adherent to the diverticular walls.

These diverticula had smooth muscle coats and were lined by mucosa resembling some portion of the alimentary tract. In our first case the lining was similar to that of jejunum, colon and the respiratory tract, while in the other two it resembled gastric mucosa, it being found to secrete hydrochloric acid and pepsin.

The symptomatology in these patients was referable to: (1) The accumulation of gas or material within the thoracic portion of the diverticulum in such a way that it gave cardio-respiratory disturbances. (2) The formation of hydrochloric acid and pepsin within the diverticulum, which produced ulceration in the neck of the diverticulum or nearby intestine, so that serious hemorrhage ensued from the alimentary tract. (3) The accumulation of very acid material within the diverticulum, which produced an intense reaction in its wall and in regional structures, so that chest pain appeared. In our first subject, a child of three months, there had been bouts of dyspnea, cyanosis and vomiting, which were presumably induced by the backing up of intestinal material and gas into the long arm in the chest in a way that led to pressure on important intra-thoracic viscera. In our second patient, the huge diverticulum must have produced enormous amounts of gastric juice (the pH of which was shown to be 3.9); the discharge of this unneutralized fluid into the jejunum led to intestinal ulceration and exsanguinating hemorrhage. In our third patient, the primary complaints were related to pain in the chest, which was particularly aggravated during deglutition. This pouch was lined by gastric mucosa and was filled with a fluid containing hydrochloric acid and pepsin. This activated material had set up an intense reaction throughout the wall of the upper half of the diverticulum, which reaction had extended into regional tissues and into the para-esophageal structures.

Roentgenologic examination of our children gave important information which focused attention upon the thoracic lesions. The findings were such that they were not diagnostic of the condition, but there were certain features

which would probably permit us to recognize (or at least suspect) this abnormality if we encounter it again. In the first patient, loops of intestine containing air and gas were seen above the diaphragm to the right of the heart shadow, which led to a diagnosis of a diaphragmatic hernia (which indeed the patient had). After surgical reduction of the diaphragmatic hernia, some air-filled loops could still be seen on the right side of the chest (which on subsequent operation were shown to be due to the presence of the elongated and coiled diverticulum which remained in this area). In all three cases, there was some sort of a mass extending out from the mediastinum, usually to the right, which had the appearance of a long, narrow structure contiguous to and running along the mediastinum, or it had a more bulbous and rounded shadow, projecting out prominently from some part of the mediastinum. Because of obscuration by the normal cardiac or mediastinal shadows, it was not always possible to visualize the entire intra-thoracic part of a narrow diverticulum. Conversely, when the diverticulum had a large diameter, it could be seen to encroach upon the pleural cavity and to extend from the diaphragm to the apex of the chest. In one instance, this mass was very large and had a central indentation (produced by the azygos vein) which gave it a dumbbell shape. The shadows were opaque if the diverticula contained fluid, but in some instances they were radio-lucent whenever gas became pocketed in the pouch. It was of value to have film studies with the patient in an upright position to detect any tiny bubbles of gas, which were missed when film exposures were taken with the subject in a supine position. In only one instance did barium pass into the diverticulum during contrast study of the alimentary tract. Solid thoracic shadows might suggest neoplasm, teratoma, or indeed a gastrogenous cyst (duplication) of the esophagus. It has been our experience that most duplications of the esophagus do not communicate with the esophagus and hence do not contain gas. Therefore, the finding of air in a cystic structure projecting from the mediastinum has tended to make us think more of a diaphragmatic hernia, or of a diverticulum of the small intestine which projects into the thorax, such as we report herewith.

The surgical treatment of these abnormalities might lead the surgeon on a long therapeutic course requiring several operative procedures, or it might be possible to remove the entire lesion at one sitting. In our first case, being somewhat nonplused by the anomaly which was discovered, more operations were undertaken than perhaps were necessary. For the first stage, an abdominal approach was made, reducing the diaphragmatic hernia and suturing the diaphragmatic edges to the diverticulum to prevent recurrence of intestinal herniation. In a second attack, the chest was opened, but little could be accomplished because of the poor status of the baby. At a third operation, the right pleural cavity was again opened and the entire portion of the diverticulum which lay within the right half of the chest was removed. At a fourth stage, the left pleural cavity was traversed and the remaining thoracic part of the diverticulum was excised. In our second patient, the entire diverticulum

(intra-thoracic as well as abdominal) was removed in one stage, employing separate exposures through the right pleural cavity and the right flank. The baby stood this extensive maneuver in a very satisfactory way. In our third patient, the entire intra-thoracic portion of the diverticulum was removed at the first stage. At the next sitting, an anterior abdominal approach was made which gave extremely poor exposure and through which it was impossible to reach the diverticulum with any degree of satisfaction. At a third operation, an approach was made through the right flank; the intra-abdominal part of the diverticulum (from the duodenum) was so short that it appeared unnecessary and indeed unwise to remove it. Our experiences have shown that it will usually be impossible (with any degree of safety) to remove the infra-diaphragmatic portion of one of these diverticula through an anterior abdominal approach. Conversely, our limited knowledge indicates that the infra-diaphragmatic portion of such a malformation can be removed in some instances through a right flank incision (splitting a portion of the diaphragm if necessary to obtain adequate exposure).

Possibly, it is not necessary to remove all of the diverticulum in each case where this malformation is found. When the symptoms are due entirely to the accumulation of fluid and gas within the intra-thoracic portion of the diverticulum, it is probably sufficient to excise only that part which lies above the diaphragm. When, however, a diverticulum is lined by gastric mucosa which secretes hydrochloric acid and pepsin so that there is severe chest pain or when there is intestinal hemorrhage, it becomes highly desirable to remove the entire diverticulum if this is technically possible.

CASE REPORTS

Case 1.—L. C., A314627, a female infant, entered The Children's Hospital at the age of 3½ months with a chief complaint of intermittent episodes of vomiting and cyanosis since the age of 8 weeks. The family history was non-contributory. Birth was at full term; the weight was 5 lbs. 4 oz. For the first 2 months of life the infant appeared to be perfectly healthy.

At the age of 2 months the patient began to regurgitate about one feeding daily. During the following week an upper respiratory infection developed, and associated with this were episodes of cyanosis, lasting from a few seconds to 5 or 10 minutes, which were relieved by the use of oxygen. During the thirteenth week severe cyanosis frequently occurred after feedings. Roentgenographic examination showed loops of intestine in the right side of the chest and a diagnosis of diaphragmatic hernia was made.

Physical examination showed a well-developed and nourished infant in no distress at the moment. The neck was short and the patient resisted efforts to move the head. The chest was of normal size and shape; the expansion was good on either side. Breath sounds were audible over both lung fields, but in the lower part of the right side of the chest occasional peristaltic gurgling sounds could be heard. The abdomen was slightly scaphoid, but was otherwise normal. The remainder of the physical examination was essentially negative.

Roentgenographic examination following entry revealed multiple congenital anomalies of the cervical and upper thoracic vertebrae of a Klippel-Feil type. Several intestinal loops were seen in the right side of the chest. Two hours following a swallow of barium,

small amounts of the material appeared in the "intestinal" loops in the right side of the chest (Fig. 1).

Believing that the baby had a right diaphragmatic hernia, an abdominal exploration was carried out on February 18, 1947. Posterior to the liver, a defect about 1.5 cm. in diameter was found in the right side of the diaphragm, this opening being situated just above the crus. Several loops of ileum were pulled down out of this diaphragmatic defect. After this was finished there remained—to the surgeon's amazement—a *single* limb of bowel running up into the chest (Fig. 2). This had the diameter, thickness and general appearance of intestine. Tracing it downward, it was found to run behind the antrum of the stomach, alongside of the superior mesenteric vessels and to arise as a T-shaped side-arm from the jejunum (about 3 inches beyond the ligament of Treitz).

FIG. 1.—(Case 1.) Preoperative roentgenogram of chest showing air filled intestinal loops in the right side of the thorax.

This diverticulum had a wide communication with the jejunum. After making these observations, attention was again turned to that portion of the diverticulum which pierced the diaphragm. Traction showed that it was securely anchored within the thoracic cavity; it was impossible to determine the extent of and the attachments of the intra-thoracic part of the structure because of the small size of the diaphragmatic orifice. It seemed much too risky to attempt removal of the abdominal portion of the diverticulum without knowing more about its intra-thoracic extension. Therefore, it was decided to leave it in place, but to tack its surface to the edges of the surrounding diaphragmatic opening to prevent a recurrence of herniation through this region (Fig. 3). Immediately following this operation, roentgenographic examination still showed barium and air-filled loops of "intestine" in the right side of the thoracic cavity. Piecing together all our bits of knowledge, it was now obvious that these structures above the diaphragm (seen by roentgenographic means) must be a long, coiled diverticulum—the lower part of which we had viewed (at operation) in the abdominal cavity.

The following day a right transpleural exploration was carried out. The lung was found to be free and was unattached to the surrounding structures. A mass, somewhat larger than a golf ball, was seen extending up from the postero-lateral portion of the diaphragm, this being covered by a thin parietal pleura which had been pushed out from the mediastinum. Upon opening this sac, there appeared to be several loops of "intestine" which were quite adherent to one another; when these were disengaged we had a

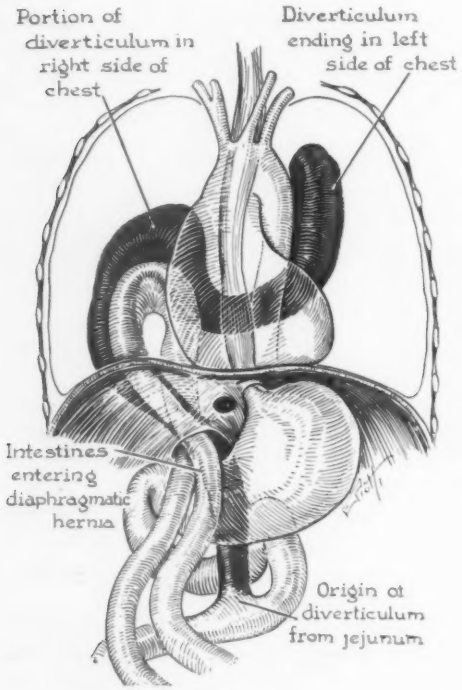
Portion of diverticulum in right side of chest
Diverticulum ending in left side of chest
Intestines entering diaphragmatic hernia
Origin of diverticulum from jejunum

FIG. 2

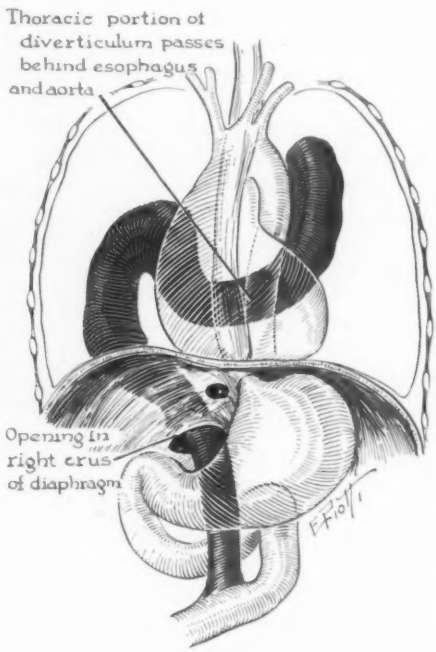

FIG. 3

FIG. 2.—(Case 1.) Sketch of findings which were gathered from roentgenologic investigations and from operations. There is a diaphragmatic defect on the baby's right. Intestinal loops had prolapsed upward through this orifice. In addition, a long diverticulum from the jejunum passed up through the diaphragmatic opening, entered the right side of the chest (extra-pleurally) and then coursed behind the esophagus and aorta to reach the left side of the chest, ending near the left apex of the thoracic cage.

FIG. 3.—(Case 1.) Status of findings *after* reduction of the diaphragmatic hernia—showing the long jejunal diverticulum extending up into the chest. Accumulation of fluid and gas in the diverticulum caused bouts of respiratory distress. In multiple stages, all of the thoracic portion of the diverticulum was removed, leaving the infra-diaphragmatic part.

tubular structure, 10 to 12 inches in length, one end of which came up through the diaphragm and the other end of which disappeared into the mediastinum! Dissection showed that its upper end went behind the esophagus and the aorta and kept on going into the left side of the chest. This tube, which was obviously a continuation of the diverticulum we had seen in the abdomen the day before, received many blood vessels from regional tissues. The baby's condition suddenly became poor and it was necessary to close the chest quickly. The liberated portion of the diverticulum was dragged out through the back of the wound.

Following this second operation, the child's condition was very precarious for several days. After the general condition had become stabilized, the exteriorized loop was opened so that barium could be injected into its two limbs. Film and fluoroscopic studies clearly showed that the diverticulum went downward to communicate with the jejunum and that its other end traversed the chest and ran up to the left of the spine to reach the left apex of the chest!

Unfortunately, the baby developed a diarrhea (which was epidemic in the ward at the time) and no further surgical attack was possible until April 8, 1947, at which time the right pleural cavity was again opened and the entire segment of diverticulum which lay in the right half of the chest was freed. It was then cut off at the diaphragm, closing that portion which ran down into the abdomen by turning it in with interrupted silk sutures. The upper end of the structure which entered the mediastinum was now cut off, so that a section about 10 inches in length could be resected and discarded. Into that part which remained and ran over into the left side of the chest, we threaded a small rubber catheter which was brought out behind the parietal pleura and passed through a stab wound in the back. This catheter was left in place to prevent any accumulation of fluid in the remnant which remained in the left side of the chest. The right lung was now re-expanded and the wound was appropriately closed. The course following this operation was uneventful.

On May 2, 1947, the remaining portion of the diverticulum was removed through a left transpleural thoracotomy. The lung was held forward and the mediastinal pleura was opened, to disclose the diverticulum coming from behind the esophagus and aorta in the middle third of the thorax. The diverticulum then took a sharp, upward bend to course along in the paravertebral gutter reaching almost to the apex of the chest. It received many blood vessels from regional structures but none of these was very large. The diverticulum could be liberated from its bed without much difficulty. The lung was re-expanded and the chest was closed. The convalescence was uneventful and the child was discharged home 12 days later.

The patient has been followed for 2 years since operation and has been completely relieved of her attacks of vomiting, cyanosis and dyspnea.

Pathologic examination of the specimen showed that it had a double coat of smooth muscle, the outer layer of which were longitudinal fibers and the inner layer of which were circular in distribution. The mucosal lining showed a variable picture and in most areas was quite similar to that of normal jejunum. In other zones the glands were more suggestive of those of colonic mucosa. In one small area the intestinal villi were covered by a pseudo stratified epithelium simulating respiratory epithelium, though they did not possess cilia.

Case 2.—R. M., A333598, a white male age 4 months, entered The Children's Hospital on August 13, 1948, with a chief complaint of pallor of 2 months duration and tarry stools of 11 days duration. The infant had been born at full term, with a birth weight of 6 lbs. 4 oz., and the early development appeared to have been quite satisfactory.

At 2 months of age the baby had increasing pallor and became increasingly irritable. The appetite became poor. At 4 months of age the child had loose, tarry stools and was hospitalized elsewhere for a period of 9 days. During this time the child was in a critical condition, the blood count falling to as low as 1.5 million and stools continuing to show evidence of great blood loss. They were frequently tarry and on multiple tests there was always a strongly positive guaiac reaction. With many transfusions the baby's general condition rapidly improved. A roentgenogram showed a large mass in the right side of the chest and the baby was sent to us for further investigation and treatment.

Physical examination revealed a fairly well-developed and nourished infant who was very pale but active and alert. His weight was 10 lbs. and 12 oz. There was a short, soft systolic murmur over the left side of the sternum. Otherwise, the heart and lungs were

normal by auscultation. The liver was palpable 2 finger-breadths below the costal margin and the spleen was easily palpable. Rectal examination revealed no masses but the stool was black and the guaiac reaction on it was strongly positive. Laboratory data revealed a hemoglobin of 10.4 Gm., a red blood count of 3,100,000 and a white blood count of 10,000. Roentgen examination showed a large dumbbell-shaped shadow extending out from the right side of the mediastinum, reaching from the diaphragm to the apex of the chest (Fig. 4). Visualized by a swallow of barium, the esophagus was seen displaced somewhat forward by the mass which extended into the posterior mediastinum. No barium flowed into the mass, but on several films bubbles of air could be seen within it, indicating that it probably possessed some sort of a communication with the alimentary

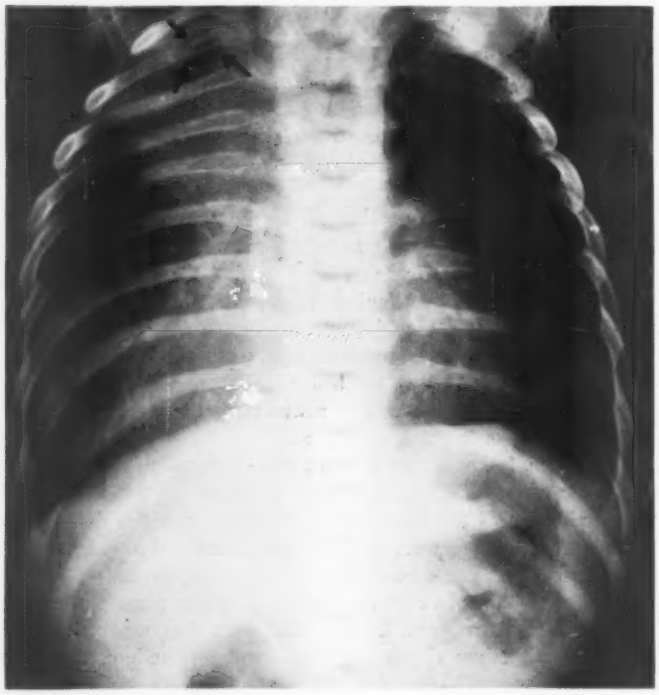

FIG. 4.—(Case 2.) Preoperative roentgenogram showing the large diverticulum projecting out from the right side of the mediastinum. Arrows indicate gas bubble in the structure. (The central groove or depression was caused by the azygos vein which constricted the diverticulum).

tract. No abnormalities were detected in the stomach, duodenum, or jejunum. There were developmental anomalies in the lower cervical and in the first two thoracic vertebrae.

During a period of a week, the baby required numerous transfusions to overcome the effects of continuing, massive bleeding from the gastro-intestinal tract. On August 20, 1948, a right, transpleural thoracic exploration was carried out. Behind the lung, and extending throughout the entire length of the chest, a cystic mass bulged out from the mediastinum; the middle portion of this was constricted by the azygos vein. It was covered by mediastinal pleura, which was now opened. The exposed structure was a thick-walled sac containing fluid and gas quite adherent to the apex of the chest.

It narrowed down inferiorly to a neck which had all the appearances of intestine as it pierced and ran through a defect in the posterior part of the diaphragm. With very little difficulty this entire structure was freed from its bed in the thorax. Inasmuch as the child appeared to be in reasonably good condition, it was decided to open the abdomen and examine the lower end of the diverticulum. A subcostal abdominal incision was made, running from the posterior axillary line slightly downward and well forward to the lateral border of the right rectus muscle. The liver was pushed forward and an

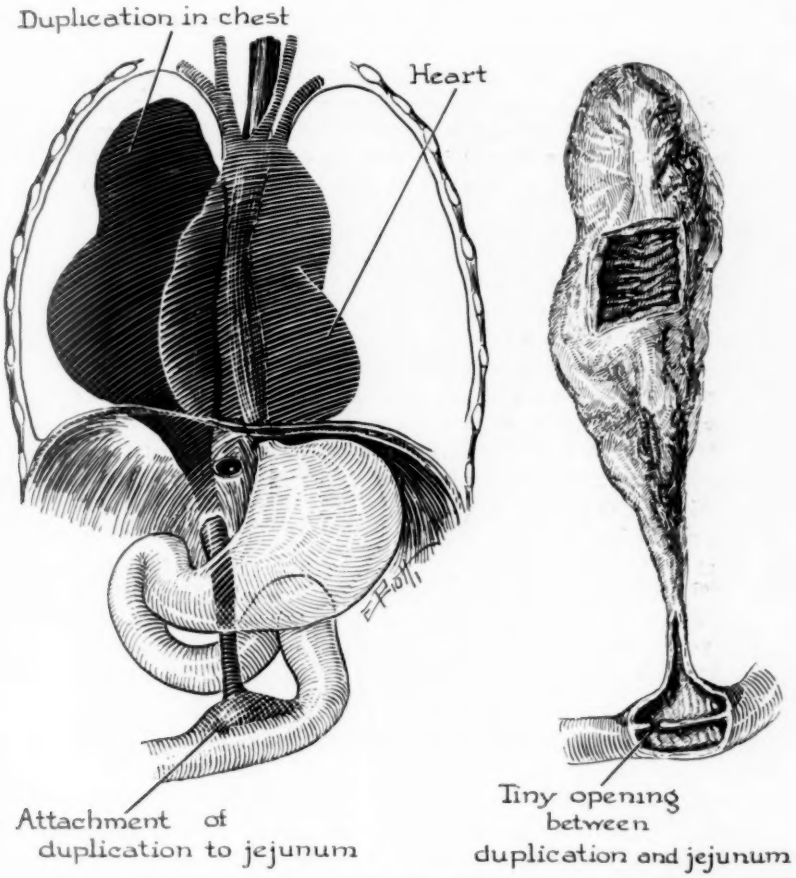

FIG. 5.—(Case 2.) *Left:* Sketch of large jejunal diverticulum which extended up into the right side of the thorax. The diverticulum was lined by gastric mucosa; its acid secretion caused jejunal ulceration and severe hemorrhage. The entire anomaly was excised by a combined transpleural and a transabdominal one-stage procedure. *Right:* Sketch of the specimen and segment of jejunum which were removed.

excellent view was obtained of the diverticulum which came down through the right crus of the diaphragm, coursed in front of the pancreas and then joined the jejunum about 2.5 cm. beyond the ligament of Treitz (Fig. 5). This diverticulum was freed from its attachments to the surrounding structures and was also cut away from the ring where it pierced the diaphragm. A segment of jejunum (several centimeters in length) which gave

rise to the diverticulum was resected, and the continuity of the jejunum was re-established by an end-to-end reconstruction. At this stage the entire diverticulum, including its thoracic portion, its intra-abdominal part, and the segment of jejunum from which it arose, could be discarded. The defect in the diaphragm was quickly repaired. The lung was re-expanded and the chest wound was closed. Finally, the abdominal wound was closed.

The postoperative course was uneventful, convalescence was rapid, and the patient was discharged from the hospital on the eleventh postoperative day.

Pathologic examination of the specimen showed it to have a double coat of smooth muscle, the outer longitudinal layer of which was quite thin, although the inner circular layer was well developed. The lamina propria was similar to that of normal bowel. In one section there was an island of pancreatic tissue lying in the lamina propria. The mucosa showed a variable picture. In one section there was some tissue which resembled duodenal mucosa. For the most part the lining, while not entirely characteristic, was most suggestive of a gastric type of lining. The fluid within the diverticulum had a pH of 3.9. The duplication communicated with the jejunum through an opening about 0.5 cm. in diameter.

Case 3.—R. W., A341531, a 4-year-old white male, entered The Children's Hospital with a chief complaint of pain in the middle of the back, which had been aggravated by eating and which had been present for about one year. Birth was at full term, with a breech delivery. Birth weight was 6 lbs. 7 oz.

The baby had presented a feeding problem for the first few months of life and had continued to spit up food until the age of 2½ years. One year prior to entry in this hospital he began to complain of pain in the back, especially during and following deglutition. This pain was most intense between the scapulae; it became more frequent in appearance, and sometimes caused bitter complaints two or three times during a day. The pain was often accompanied by pallor and sweating.

Physical examination revealed a well developed and nourished white male, who was in no distress at the time of examination. There appeared to be slight mental retardation. A grade II systolic murmur was noted over the entire precordium, being more intense over the pulmonary area. There were persistent ronchi in the lower one-half of the right lung field anteriorly. The liver edge was palpable to the costal margin. The remainder of the physical examination was essentially negative. Laboratory data revealed a normal urinalysis, hemoglobin of 13.7 Gm., a red blood count of 4.85 million, a white blood count of 8,500, a negative Hinton test, and a negative tuberculin test (1-1000). Film and fluoroscopic studies showed nothing unusual in the trachea or in the upper mediastinal shadows. The heart was apparently within normal limits of size, shape, and position. Along the lateral and posterior aspects of the heart on the right side there was a well-defined, soft-tissue mass which paralleled the heart border (Fig. 6). No pulsations were noted in this mass, and no definite alteration in its contour could be noted when the patient was placed in different positions. The barium-filled esophagus showed a minimal deformity in its lower third; its right border was slightly compressed.

Exploration of the right pleural cavity was carried out on February 21, 1949. The lung was held forward, and a long, sub-pleural, tubular structure was found, running along the mediastinum, extending from diaphragm almost to the apex of the chest. As this came up through the right crus of the diaphragm it had the size, thickness and general appearance of intestine. In its mid-portion, it ballooned out to 8 to 10 cm. in diameter, had a thicker wall and was quite adherent to surrounding viscera. In its upper third, its wall was several times the thickness of intestine, was very congested and vascular, and had dense, hemorrhagic adhesions to the esophagus and regional structures. (This reaction had come from the unneutralized hydrochloric acid and pepsin within its lumen).

Though the dissection was very difficult, the entire intra-thoracic part of the diverticulum was excised. A catheter was led into the infra-diaphragmatic part of the diverticulum and this tube was brought out through a stab wound in the back of the thorax. The right lung was now re-expanded and the chest was closed appropriately. From this operation the child made a rapid and satisfactory convalescence. A thin barium mixture was injected through the catheter and it was obvious that there was some sort of a connection with the upper part of the intestine, but the exact location of this union was difficult to determine. The catheter was then pulled out.

Believing that an infra-diaphragmatic segment of considerable length might still remain and that it was desirable to remove it, the abdomen was explored on March 4, 1949. A vertical opening was made through the upper part of the rectus muscle, but a totally inadequate exposure was obtained. The diverticulum could be identified coming through the right crus of the diaphragm, but since it was tucked in behind the vena cava and the gastrohepatic ligament it was utterly impossible to view it completely and trace it adequately. The examination was especially unsatisfactory because the pancreas and the

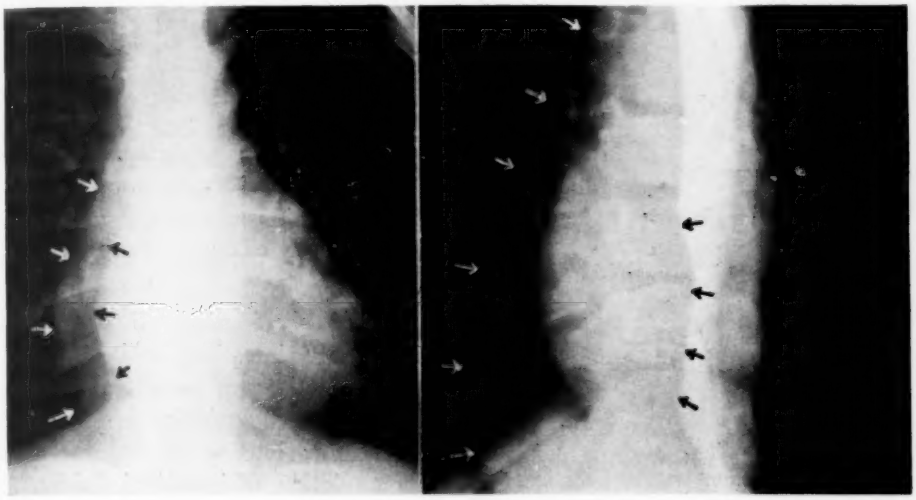

FIG. 6.—(Case 3.) *Left:* Roentgenogram of the chest, showing mass protruding from the right side of the mediastinum (right border of heart indicated by black arrows. Wall of diverticulum shown by white arrows). *Right:* Heavier roentgenographic exposure, with barium in the esophagus, showing the thoracic mass pressing upon the esophagus (arrows outline borders of the diverticulum).

entire duodenum were apparently displaced upward against the under surface of the liver in an abnormal fashion. Because of the difficulties encountered, the exploration was abandoned.

A second abdominal operation was carried out on March 15, 1949; with the boy turned well up on his side, an incision was made in the costovertebral angle, running downward and forward, and the tenth, twelfth and eleventh ribs were removed. The liver could be displaced forward and the crus of the diaphragm viewed fairly well. The diverticulum which came through it was quite short and, within one or two centimeters, communicated widely with the posterior surface of the first part of the duodenum (Fig. 7) which had been drawn up against the diaphragm by this anchoring attachment. After carefully evaluating the situation, it seemed that the risks of removing so short a diverticular stump far outweighed the possible benefits of such an excision. It was therefore decided to

leave this remnant, believing that its wide communication with the duodenum would allow reflux of duodenal contents into it in such a way as to neutralize any acid which it might be secreting. Following this exploration the child made a rapid recovery and was discharged 8 days later. He was completely relieved of the chest pain for which he had entered the hospital.

Pathologic examination of the specimen showed that it had a two-layered coat of smooth muscle. In some sections there appeared to be an additional irregular third coat of smooth muscle. The muscularis mucosae was well defined. There was a wide submucosa of loosely arranged tissue, containing islands of fatty substance. The lining was quite similar to that of a normal gastric mucosa. Numerous acid-secreting cells were found throughout the specimen.

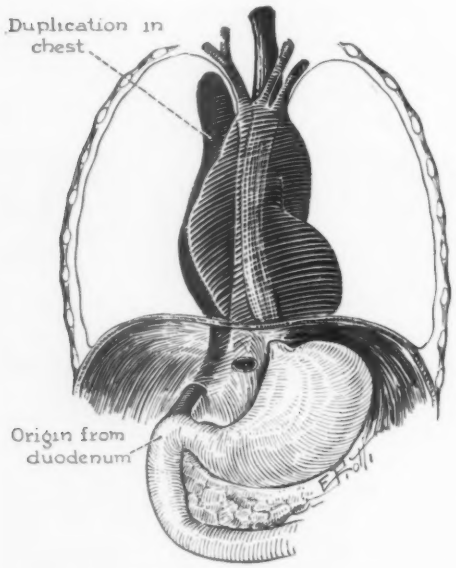

FIG. 7.—(Case 3.) Sketch of findings of duodenal duplication with a long extension into the chest. The diverticulum was lined by gastric mucosa which secreted a fluid containing hydrochloric acid and pepsin. This unneutralized material set up an intense reaction in the diverticular wall and adjacent mediastinum so that the child had severe pain during deglutition. All of the thoracic part of the diverticulum was removed. The short infra-diaphragmatic part was not excised.

SUMMARY

Descriptions are made from three children who had unusual malformations in the form of long, duodenal or jejunal diverticula which coursed upward, pierced the right side of the diaphragm, and extended far up into the chest. These elongated or globular, hollow structures had smooth muscle coats and were lined by mucosa similar to that of some portion of the alimentary tract. In one of them the lining was histologically like that of the upper intestine, while in the other two the mucosa was gastric in type. The diverticula gave symptoms in various ways. In one, the accumulation of intestinal fluid and gas within the intra-thoracic portion of the structure gave pressure on thoracic organs and led to intermittent dyspnea and cyanosis. In the second case, the formation of large amounts of gastric juice within the diverticulum produced ulceration in the jejunum from which there was hemorrhage of almost exsanguinating degree. In a third patient, the pocketing of unneutralized gastric juice within the sac brought on inflammatory changes within its wall and reaction in surrounding structures which resulted in chest pain. Roentgenologic examination of these three patients showed variable features, but in each there was a shadow projecting out from the mediastinum which appeared to be either a solid mass or a gas-containing viscus.

Under some circumstances, the symptoms from one of these diverticula are such that excision of the entire malformation is desirable, but in other

cases complete relief can be obtained by removing only the thoracic portion of the diverticulum.

Surgical attack on such an anomaly might require multiple-stage procedures, depending upon the extent of the diverticulum, the condition of the subject, and the appropriateness of the operative exposure which has been chosen. It is sometimes possible to remove the intra-thoracic and the infra-diaphragmatic portions of the diverticulum in one operation.

INTRACAPSULAR FRACTURE OF THE NECK OF THE FEMUR;

OBSERVATIONS ON 152 PATIENTS TREATED BY INTERNAL FIXATION WITH THE SMITH-PETERSON NAIL*

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THE FOLLOWING OBSERVATIONS and conclusions are based upon a study of 152 consecutive patients with intracapsular fractures of the neck of the femur treated from January, 1937, to January, 1948. All of the cases were treated by Smith-Peterson nail fixation, and all deaths or complications that occurred during the hospital stay are included.

No comments are needed to emphasize the great improvement in results with the use of internal fixation over the older methods of treatment. Of the various types of internal fixation we feel that the Smith-Peterson nail is the ideal, in that it produces firm fixation, at the same time preventing rotation of the fragments. We do not advocate the use of any of the screw methods for this purpose as we feel that the screw threads will not keep a prolonged firm hold in the intertrochanteric or neck region of the bone. As a result, loosening of the screw may occur and rotation and motion of the neck fragment on the head may follow, with resulting non-union. We have seen this happen in two instances.

Vitallium nails are used exclusively. In a few cases where the nails were removed at the end of one or two years, no change in the appearance of the nail could be observed. We have had only one nail break and that was where the patient slipped from a height of two feet, landing without support on the injured limb. As he was a heavy person the same result would probably have happened with any other metal. There have been only a small number of nails which have loosened and become extruded, and it is felt that this is due in no small part to the absence of ionization, with bone necrosis around the nail.

We have used internal fixation in this fracture regardless of the age of the patient. It is our feeling that if those patients in the advanced age group do not survive the operation, they would not have survived very long without it. As a matter of fact, it is surprising how well they stand this procedure. There seems to be very little shock involved and the physical resistance of the patients is apparently greater than is frequently anticipated. This may also be noted in other operations in the aged. Their vitality and general cardio-renal circulatory status is undoubtedly usually good to begin with, as otherwise they would not have lived to such an advanced age. In the winter of 1946–1947 we operated upon 20 patients over 80 years of age for this fracture with only

* Submitted for publication March, 1949.

two postoperative deaths. The 18 patients who survived did very well postoperatively and left the hospital in good physical condition. As Watson-Jones has said, "An expectation of life amounting to no more than five or ten years is no justification for the miseries of the un-united fracture; the old have as much right to live in comfort as the young."

We do not nail these hips as an emergency procedure. We sometimes wait for as long as a week, allowing the patients to get over the shock of the injury and in the older group to permit their general physical status to be evaluated. During this interval the limb is suspended with Russell traction, using 10 lbs. weight. In this period of Russell traction we have found that the fracture is practically always reduced, making any reduction in the operating room unnecessary.

Kirschner wires as a preliminary guiding step in driving the nail are not used as it is felt that with our technic, driving the nail over a Kirschner wire is unnecessary. There have been cases reported, where with this method, the distal portion of the wire was sheared off by the nail, and this portion of the wire forced by the nail up into the pelvis. Mechanical aids for directing the course of the nail are not used, as they seem to be unnecessary and frequently unreliable.

Our routine for treating these patients and the technic of nailing may be outlined as follows:

1. The extremity is placed in Russell traction with 10 lbs. weight for a period up to seven or eight days. During this time the patient's general condition is evaluated. Any cardiac or renal disease is treated and other conditions, such as diabetes, are brought under control. The patient's fluid balance is cared for and intravenous fluids or blood are used as indicated.

2. Forty-eight hours before operation the skin of the thigh, groin and abdomen are prepared by shaving, scrubbing with soap and water, cleansing with alcohol, and encasement in sterile drapes. This skin preparation, except for shaving, is repeated 24 hours later. The sterile drapes are then left on, being removed in the operating room. We believe that this preliminary skin preparation helps to diminish the chances of infection.

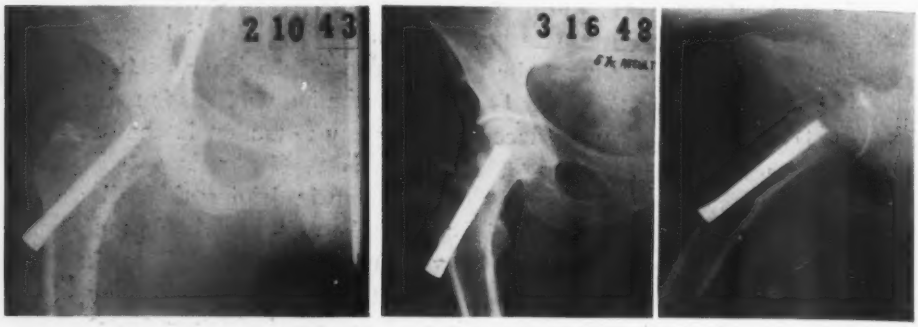
3. On the day of operation the Russell traction is removed, and a Thomas leg splint is applied, with traction maintained to the distal end of the splint. This is done to continue the traction while the patient is being transported to the operating room and until anesthesia is induced.

4. Our choice of anesthesia varies with the individual patient but we most frequently use avertin and gas oxygen, pentothal and gas oxygen, or pentothal and curare. Spinal anesthesia is not used. A roentgen ray cassette holder is placed under the patient before the anesthesia is started.

5. After the patient is anesthetized, the Thomas splint is removed and an assistant holds the extremity at the foot and lower leg with the knee in full extension and no abduction at the hip. *The extremity is internally rotated about 15 degrees and this position is maintained throughout the operation. This 15 degrees internal rotation brings the neck of the femur to a position*

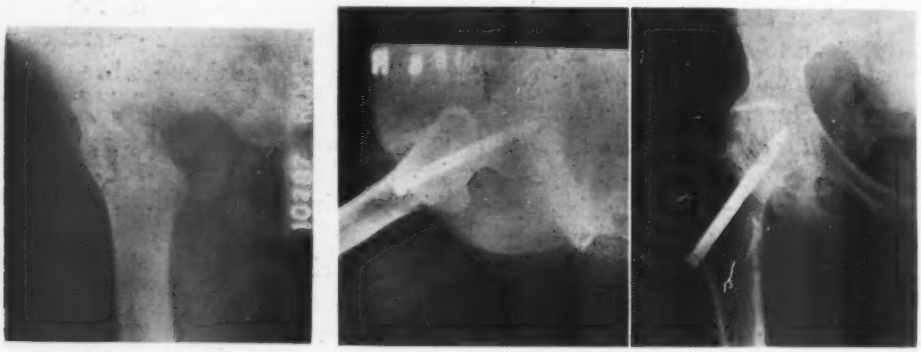
parallel with the table or the floor and it is important that this position not be allowed to change.

6. A towel clip is then placed on the skin of the abdomen halfway between the antero-superior spine of the ileum and the spine of the pubis. This point is usually directly over the head of the femur. Antero-posterior and lateral roentgenograms are then taken to be sure that there is a good reduction and to



A B

FIG. 1.—(Case 1, A) Subcapital fracture in a 59-year-old woman at the time of nailing. A towel clip is in position on skin centered over head of femur. (B) The same patient at end of five years, showing good bony union and no absorption of the neck.

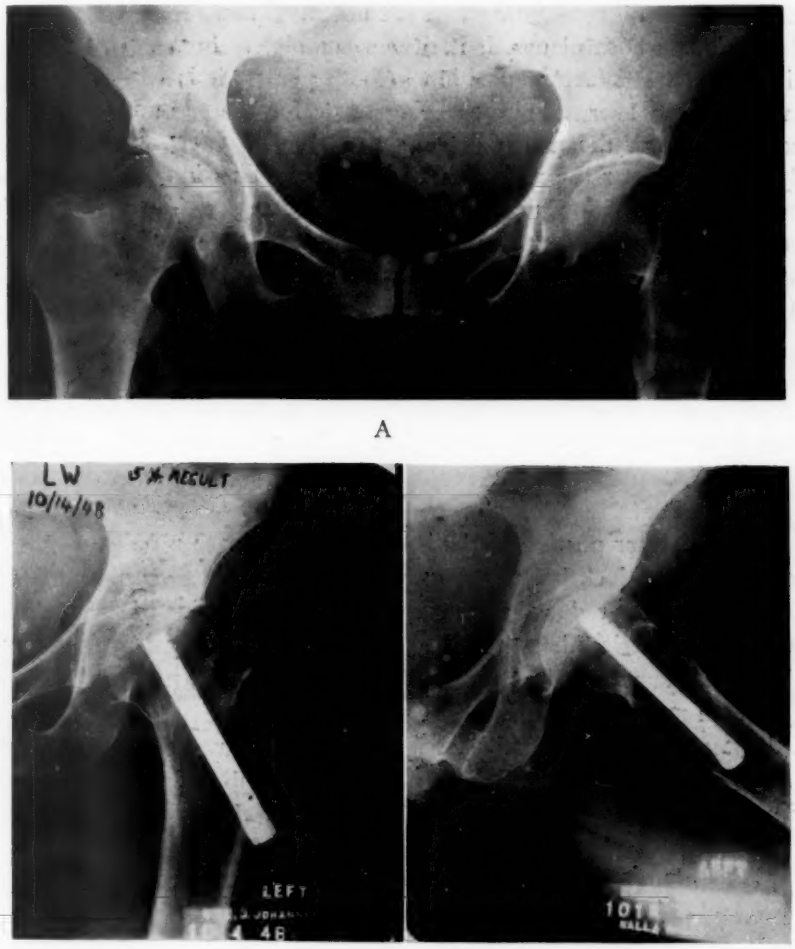


A B

FIG. 2.—(Case 2, A) Subcapital fracture in a 73-year-old woman. (B) Eight-year result showing good bony union.

check the position of the towel clip. If the reduction is not complete, a Leadbetter maneuver is performed, although, with use of preliminary Russell traction we have usually found a complete reduction has resulted. The skin is now prepared and the operative area draped. The original towel clip is removed just before the painting of the skin. A second sterile towel clip is applied so that its position is directly over the center of the head of the femur. This may be at the same point as the original towel clip or a little higher or lower, depending upon the position of the original towel clip as seen in the antero-posterior roentgenogram.

7. A longitudinal incision about 6 inches in length is then made on the outer side of the thigh extending downwards from the greater trochanter. The fascia and muscles are split and the outer side of the shaft of the femur and the greater trochanter are exposed by sharp and blunt dissection. At a point one inch below the greater trochanter a hole is driven with a bone punch



A

B

FIG. 3.—(Case 5, A) Fracture of the neck of the femur in a 60-year-old woman. Date of fracture is September 25, 1943. (B) Same patient October 14, 1948. Five-year result showing good bony union and no absorption of the neck.

into the outer aspect of the shaft of the femur midway between the anterior and posterior surfaces of the bone. This hole is enlarged to take the end of the nail. The driving of the nail is then started. It is directed towards the towel clip in the antero-posterior plane and is kept exactly parallel with the

operating table or floor in the lateral plane. An assistant should watch carefully to see that this parallel position of the nail is maintained all of the time the insertion of the nail is taking place, and care should also be taken to make sure that the other assistant holding the extremity is keeping it *in 15 degrees internal rotation*. Before the nail is completely driven in, the operation is halted, the field is covered with a sterile sheet and another set of antero-posterior and lateral roentgenograms are taken. If these show the nail to be in good position in both planes, it is driven completely home. If, however, the nail is not in a satisfactory position in both planes, it is withdrawn and the nailing is started over, allowance being made for the error in position noted in the films. After the nail is driven home, the fragments are carefully and *firmly impacted* over the nail with an impactor. This impaction of the fragments should be vigorous, and it is in our opinion a very important step in the procedure. It should never be omitted. It has been our experience that any case where light can be seen between the fragments in the postoperative roentgenograms, is almost surely doomed to go on to non-union.

8. The length of the nail to be used may be estimated before operation by taking an antero-posterior roentgenogram of the hip in traction with a one-inch lead marker placed longitudinally on the skin of the outer side of the thigh over the greater trochanter. This marker is then measured on the film, and as some magnification is always present in roentgenograms, it will usually be found to measure from 1⅛ inches to 1¼ inches. The distance from the joint surface of the center portion of the head of the femur to a point on the outer side of the shaft 1 inch below the greater trochanter is then measured. This measurement is then reduced according to the magnification of the lead marker and will give the desired length of the nail to be used. In most patients we have found that the female patient requires a 3½ to 3¾ inch nail, and the male a 3¾ to 4 inch nail.

9. Postoperatively the patient is given penicillin routinely for about one week. No suspension of the limb is used. Early ambulation is urged and we attempt to have these patients out of bed in a wheelchair within a few days. This may vary between one day and one week, depending upon the age and strength of the patient. An ambulatory Thomas splint is then made for each patient, as unsupported weight bearing is not allowed for six months or until roentgenograms clearly show bone trabeculations crossing the fracture line.

In this series there were 94 women and 58 men. The ages varied from 28 years to 89 years, with the large majority over 65 years of age.

The age distribution by decades was as follows:

Age	Number of Patients	Age	Number of Patients	Age	Number of Patients
20–30	2	50–60	18	70–80	42
30–40	4	60–70	45	80–90	30
40–50	11				

There were 12 deaths, or a mortality rate of 7.8 per cent. The causes of death were as follows:

Sex	Age	Cause
Male	83	Bronchopneumonia with roentgen-ray evidence of consolidation.
Female	82	Bronchopneumonia—eleventh postoperative day.
Female	89	Generalized artero-sclerosis. Cardiac decompensation. Uremia.
Female	80	Uremia.
Female	65	Sudden dyspnea resulting in death on seventeenth postoperative day. Diagnosis: Acute myocardial or pulmonary infarction.
Male	82	Wound infection. Bilateral bronchopneumonia (autopsy).
Male	58	Patient had tabes dorsalis and cord bladder with pyelonephritis on admission; associated supracondylar fracture of the opposite femur. Non-protein nitrogen on admission was 158. Died postoperatively of pyelonephritis and uremia.
Female	73	Acute pulmonary embolism.
Female	78	The patient was admitted with a diagnosis of generalized artero-sclerosis with cerebral degeneration. There was cachexia, a steady downhill course, and severe rectal hemorrhage of undetermined cause the day before death.
Male	76	The patient was hemiplegic on admission, also had lues and chronic bronchitis and arterio-sclerotic heart disease. His cardiac condition became progressively worse and patient died on the eleventh postoperative day.
Female	54	Extensive infection, died of septicemia.
Female	76	Sudden death two hours postoperative. The patient was a known cardiac for years and she never reacted from the anesthesia.

There were five infections or an infection rate of 3.2 per cent. Two of these patients died, one from septicemia and one from bronchopneumonia. The other three survived, but went on to non-union.

One hundred and forty patients survived the operation and left the hospital. Of this number 34 patients could not be followed after they left the hospital for a long enough period to determine whether they obtained union or not, leaving 106 patients who were followed from one to eight years. Of these only 20 patients could be followed for five years or longer. This small five year follow-up in a city hospital is due to the fact that many of these patients live in rooming houses and municipal lodging houses, and belong to a floating type of population, especially in New York. Also many of them are in the advanced age group and die before the five year follow-up period is reached.

Of the 106 patients who could be followed for at least a year, 79 patients, or 74½ per cent obtained union, verified by roentgenray, and were walking without support.

In deciding the question of bony union it is felt, as stated previously, that trabeculations must be seen crossing the fracture line before one can state definitely that bony union is present. This may frequently require the use of a magnifying glass and it is important that roentgenograms so interpreted should be taken at right angles to the fracture line, preferably in both the antero-posterior and lateral planes. Roentgenograms taken from an oblique position will distort the fracture line and may give a wrong impression due to overlapping of the fragments in such a film.

In this series the occurrence of late aseptic necrosis of the head of the femur has been very infrequent. Of the cases followed for over three years, there have been only two patients who had this complication. The frequency of this change has varied considerably in different reports in the literature and there seems to be such a wide variation in different series that a reasonable

A

B

FIG. 4. (Case 6, A) A 62-year-old male with a subcapital fracture of the neck of the left femur. Fracture treated elsewhere with a Lorenz screw; poor reduction with motion at fracture site. (B) Same patient. Lorenz screw removed and fractured neck treated with a Smith-Peterson nail six weeks after injury. Above x-rays show result at four and one-half years, with good bony union and no shortening of the neck.

explanation is difficult to arrive at. We have a feeling that incomplete reduction, inadequate impaction of the fragments, or too early unsupported weight bearing may be predisposing factors leading to later changes in the head of the bone. It is also our impression that when aseptic necrosis of the head occurs, it usually appears fairly early, that is, within the first two to two and one-half

years. Changes at a later date may usually be traced back if earlier roentgenograms are carefully reviewed.

As Engel and May have shown, new blood vessels will spread up from the neck into the head along the path of the nail. They have demonstrated this in sections taken from the bone along the nail, in patients who have died at the end of one or two months. As late aseptic changes in the head are apparently due to deficient blood supply, any method of increasing the blood supply is desirable. It can thus be seen that the nail should extend well into the head and that the fracture surfaces should be in close contact. This is best accomplished by real impaction as noted above.

In attempting to evaluate the causes of non-union in this fracture we feel the following criteria are of importance:

1. A good reduction is essential.

2. The nail should be long enough to reach well into the head. A nail that is short and that does not have a good bite in the head will frequently come loose as time goes on.

3. The fragments must be firmly and completely impacted after the nail has been driven home so that there is no space between them. We can look back upon several cases where impaction was not sufficient and where light could be seen through the fracture line in the postoperative roentgenograms. Most of these cases did not unite.

4. Lateral and antero-posterior roentgenograms at the time of nailing are essential, and the former must be clear enough to be sure that the nail is entering the head properly in this plane. This is mentioned because of the great difficulty at times in obtaining good lateral views in the obese patient in the operating room. They are an essential part of the procedure.

5. Unsupported weight bearing under six months should not be permitted, or at least not until there is roentgenray evidence of solid bony union. Trabeculations must be seen crossing the fracture line in the roentgenograms before one can safely assume that bony union is complete. Early unsupported weight bearing causes a shearing action at the fracture site and, especially in the heavy patient, tends to delay union.

6. We feel that these cases should preferably not be nailed by the general hospital staff but by a few men in each hospital who are interested and experienced in this technic. There are many details even in the technic of the nailing itself which are learned only by experience, and we have found that when the procedure is constantly in the hands of a small group the end results are much better.

7. Finally, even under the best auspices, there are still a number of these fractured necks of the femur that will not unite. This is probably due in some instances to infolding of the capsule between the fragments, preventing bone contact and thus preventing union.

CONCLUSIONS

1. One hundred and fifty-two consecutive patients with intracapsular fractures of the neck of the femur treated by internal fixation with the Smith-Peterson nail are reported. There were 94 women and 58 men.

2. There were 12 hospital deaths, or a mortality of 7.8 per cent.

3. There were five infections, an infection rate of 3.2 per cent.

4. It was possible to follow 106 patients for over one year, but only 20 patients could be followed for over five years.

5. Seventy-nine of the 106 patients followed obtained bony unions, or 74.5 per cent.

6. Late aseptic necrosis of the head of the femur was infrequent, occurring in only two instances.

7. Criteria as to the cause of non-union in this fracture are enumerated.

INTESTINAL OBSTRUCTION*

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INTESTINAL OBSTRUCTION is an important cause of hospital mortality, as shown by the fact that it is responsible for approximately ten per cent of all deaths on the surgical service at the University of Virginia Hospital. While the mortality rate to be presented is still high, recent developments directed toward the more complete protection of the surgical patient as a whole from the effects of disease and operation have markedly reduced it. The purposes of the present report are (1) to re-emphasize certain well-recognized features of the disease, (2) to mention some of the factors responsible for the encouraging improvement in mortality, (3) to discuss briefly a few of the controversial issues relative to treatment, and (4) finally, to add a group of 406 cases to the literature.

LITERATURE

The extent of the decline in the mortality of intestinal obstruction is demonstrated in Table I in which are presented results reported by several authors. Although certain differences in the mortality rates reported are due to differences in classification and inclusion of material, a striking average improvement in results is indicated.

Lehman and Becker[6] recently reported the over-all mortality in 20,137 cases cared for by the Department of Surgery and Gynecology of the University of Virginia Hospital over a period of 12 years (1934 to 1945, inclusive). A comparison of this over-all mortality with that of intestinal obstruction was made in two six-year halves of the same period. The percentage decline in mortality in the two groups of cases proved to be almost identical. The improvements in mortality of all surgical disease and in intestinal obstruction specifically were ascribed to: "(1) An increase in the accuracy of the diagnosis of disease and particularly of the diagnosis of surgical complications, such as thrombophlebitis and shock. (2) A stricter attention to, and a more accurate management of water, electrolyte, protein, and vitamin balances in the surgical patient. (3) A better understanding of the role of blood volume

* Read before the Congress of the Pan-Pacific Surgical Association, Honolulu, T. H., September 9, 1948. Submitted for publication May, 1949.

and its control. (4) Major developments in the field of anesthesia and in the mechanical control of respiratory complications. (5) The introduction of specific chemotherapeutic and antibiotic substances. (6) The adoption of certain special therapeutic measures in common surgical diseases, such as those developed for the treatment of phlebothrombosis, thrombophlebitis, jaundice, intestinal obstruction, and others."[6] The comparison presented suggests that the decline in mortality of intestinal obstruction is more largely due to general protective measures than to specific innovations in the treatment of obstruction to which most authors ascribe it.

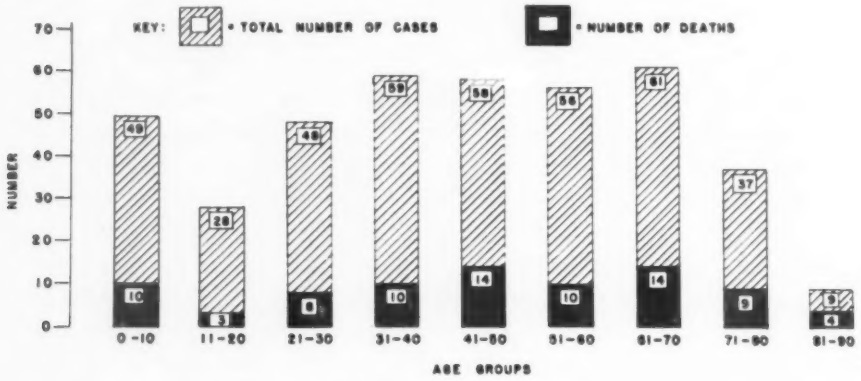

FIG. 1.—Distribution and mortality by age. Patients over 50 years of age comprise 40% of the material and 45% of the deaths. The death rate of patients up to and including 50 years of age is 18.6%. Above 50 years it is 22.7%. In one fatal case the age is not recorded.

TABLE I.—*Intestinal Obstruction Results Reported by Other Authors.*

Author	Years of Report	Number of Cases	Mortality (Percent)
Van Beuren and Smith[1]	1916–1919	60	66.6
Miller, C. Jeff[2]	1925–1929	343	60.9
Moss and McFetridge[3]	1930–1932	340	31.7
Calihan, Kennedy and Blain[4]	1936–1945	204	20.0
Eliason and Welty[5]	1934–1943	292	11.0
Becker, Davis and Lehman	1933–1947	406	20.4

MATERIAL

A series of 406 cases is reported, including all cases, with exceptions noted below, in which the diagnosis of mechanical intestinal obstruction was made on the Surgical and Gynecological service of the University of Virginia Hospital over a period of 15 years (1933 to 1947, inclusive). Excluded from the study were all cases of intestinal obstruction due to malfunctioning colostomy, rectal stricture, congenital atresia, congenital megacolon and impaction of feces. In addition, tumors of the bowel without clinical symptoms of obstruction indicating the need for prompt relief were not included. The

reported cases either were admitted with a primary diagnosis of obstruction or developed it as a complication during hospitalization.

The age distribution by decades is presented in Figure 1. The youngest patient was one day old, the oldest 86 years. The average age was 42.3 years. The age of one patient who died was not recorded.

Sixty-two per cent of the patients were males, and 38 per cent were females (Table II). Had cases of rectal stricture on the basis of lymphopathia venereum been included, the sex difference undoubtedly would have been decreased.

TABLE II.—*Intestinal Obstruction Material and Results.*

	Number of Cases	Percent of Cases	Number of Deaths	Mortality (percent)
Total Cases	406	100.0	83	20.4
Sex				
Male	253	62.3	40	15.8
Female	153	37.7	43	28.4
Site of Obstruction				
Small bowel	365	89.9	74	20.3
Large bowel	41	10.1	9	21.9
Degree of Obstruction				
Partial	137	33.7	14	10.2
Complete	269	66.3	69	25.6
Type of Obstruction				
Simple	230	56.7	40	17.4
Strangulated	176	43.3	43	24.4
Bowel Resection				
Yes	49	12.0	12	24.5
No	357	88.0	71	19.8
Duration of Symptoms				
Less than 24 hours	157	38.6	16	10.2
More than 24 hours	249	61.4	67	26.9
Etiology of Obstruction				
Adhesions	186	45.8	34	18.2
External hernia	123	30.3	17	13.8
1. Inguinal	83	20.4	9	10.8
2. Femoral	26	6.4	6	23.0
3. Others	14	3.4	2	14.3
Neoplasms	35	8.6	7	20.0
Intussusception	27	6.6	10	37.0
Volvulus	12	2.9	2	16.6
Thrombosis and embolism	8	2.0	7	87.5
Others	15	3.7	6	40.0

Since large bowel obstruction is frequently complicated by small bowel obstruction, it is difficult to classify the material in a clear-cut way between the two levels. The difficulty is increased by the fact that there are also cases of primary mixed obstruction, such as ileocecal intussusception and incarcerated hernia sacs containing both large and small bowel. Since the risk associated with these lesions is primarily that of the small bowel obstruction, mixed obstructions have been classified in that category. Obstructions limited to the small bowel and those occasional primarily mixed types accounted

for 89.9 per cent of the entire material (365 cases) (Table II). Cases of primary large bowel obstruction accounted for 10.1 per cent (41 cases).

Complete obstruction existed in 33.7 per cent of the cases and partial obstruction in 66.3 per cent (Table II).

Forty-three per cent of the patients were found to have strangulated obstruction at operation or autopsy. The line between simple and strangulated obstruction is not always easy to define pathologically. In this study all cases which showed any evidence of compromise of the circulation, no matter how slowly or how promptly relieved by the release of pressure, have been classified as strangulated obstruction. The higher incidence of strangulation in this series as compared to many reported series is probably due largely to this method of classification. Another factor is the relatively high incidence of delayed treatment due to the rural origin of the majority of the patients.

Resection of bowel was performed in only 12.0 per cent of the cases (Table II). Nine cases were admitted in a moribund condition and died before operation could be performed.

Two hundred and forty-nine (61.4 per cent) of the patients had had symptoms of intestinal obstruction for 24 hours or more before their admission to the hospital, whereas the remainder (38.6 per cent) were earlier cases (Table II).

As in most reports, the most common causes of obstruction (Table II) were adhesions and external hernia, accounting together for three-fourths (76.1 per cent) of the entire series. The incidence of neoplasms is relatively low (8.6 per cent) when compared with other reported series. In all probability this is due to the fact that in the present survey were included only those cases of tumor which had produced a degree of obstruction requiring prompt treatment. Intussusception was the cause of the obstruction in 27 cases; of these, 16 occurred below the age of ten, accounting for one-third of all obstruction in this age group. There were 12 cases of volvulus, eight cases of mesenteric thrombosis or embolism, and the remaining 15 patients were obstructed by miscellaneous lesions.

The general principles of treatment employed in this series will be outlined in the discussion.

RESULTS

In the entire series there were 83 deaths, a mortality of 20.4 per cent (Table II). When the nine patients admitted in a moribund condition are excluded from the totals, there were 397 cases with 74 deaths, a mortality of 18.6 per cent.

The major factors influencing prognosis were the degree, type, duration and etiology of the obstruction (Table II). The mortality in small bowel obstruction was 20.3 per cent; in large bowel obstruction it was 21.9 per cent. Only 10.2 per cent of the partially obstructed patients died, while the mortality of complete obstruction was 25.6 per cent. The mortality rates for the

simple and strangulated obstructions were 17.4 per cent and 24.4 per cent respectively. The relatively slight difference in these two figures is probably due to the liberal interpretation of strangulation as already described. Of the patients seen within the first 24 hours of illness only 10.2 per cent died; the mortality was almost three times as great (26.9 per cent) in the patients who had been obstructed more than 24 hours.

The mortality in inguinal hernia with obstruction was 10.8 per cent as compared with 22.9 per cent for the remainder of the cases. This type of case included 20 per cent of the total material and only 11 per cent of the deaths.

DISCUSSION

Throughout the period studied the general principle has been maintained that intestinal obstruction is a disease requiring the earliest possible surgical relief, with the exception of a small group of cases that is termed on this service "reversible obstruction." This group, it is felt, may safely and effectively be treated by non-operative measures, particularly tube decompression. It includes those cases obstructed by a subacute inflammatory process due either to recent infective peritonitis or to recent surgical invasion of the peritoneum. In most instances decompression and rest of the intestine will permit the resolution of the inflammatory process with release of fixation and diminution of edema of the bowel wall. The danger of necrosis in this group is minimal. This is the type of case often successfully treated for many decades preceding the introduction of tube decompression by simple enterostomy.

Contrary to the practice reported by some authors, it is not felt that other types of obstruction, especially those due to old adhesions, are safely treated without operation. It is generally accepted that the relief of intraluminal pressure is the primary factor in protecting the patient from death from obstruction. This implies prompt, effective decompression in all cases. The most effective tube decompression is that by a tube which has passed the pylorus. Even the most ardent proponents of non-surgical treatment admit that in a number of cases intubation beyond the stomach is unsuccessful. In most cases the interval before the tube passes the pylorus and effective decompression begins is a matter of hours, during which treatment for the most important lethal factor in the disease is limited or absent. The apparent improvement in the condition of patients during this interval is often due to the correction of physiologic disturbances which is to be undertaken in any event. Such improvement may be misleading. In the first place, therefore, intubation as a definitive method of treatment except in "reversible obstruction" entails (1) failure of an essential element of treatment in some cases, and (2) delay in such treatment in most cases.

In the second place, the danger of perforation of the bowel from necrosis can never be absolutely estimated in advance. It is relatively easy to diagnose

a massive strangulated obstruction with the usual signs of severe illness, localized tenderness, mass, fever and leukocytosis. A small localized area of necrosis cannot be so diagnosed. Too many cases of obstruction from adhesions are explored in which a single band has created one or more small pressure points of gangrene to give the surgeon much confidence in his ability to affirm in advance that there is no danger of perforation. This conception does not deny the fact that intubation and suction in many cases are safe and effective. It challenges the conception that such cases can be predicted.

For these reasons once the diagnosis of "irreversible intestinal obstruction" is made on this service, further decisions are limited (1) to the specific requirements of preoperative preparation and (2) to the time of operation. The latter is performed just as soon as the patient seems ready for it, even though decompression may seem to have been effected.

Improvements in pre- and postoperative treatment have been adopted on this service as they have developed during the years covered by this study. There is need only briefly to mention the methods now employed. All cases are given normal salt solution intravenously, immediately followed by plasma and as soon as possible by whole blood if indications for blood volume replacement continue to be present. A Levin or Miller-Abbott tube is immediately introduced and suction is continued before, during, and after operation. In view of doubts of the propriety of non-surgical decompression in supposed "irreversible obstruction" coupled with technical difficulties in its use, the Miller-Abbott tube has been employed in fewer cases than in many clinics. Chemotherapeutic drugs and antibiotics are administered wherever contamination of the peritoneum is believed to have occurred or to be imminent.

This report demonstrates once more the accepted factors that modify the mortality in intestinal obstruction. The etiology, degree, type and duration of the obstruction influence the prognosis

As in so many other diseases, promptness of treatment depending on early diagnosis is an essential factor for success. In this series obstructed inguinal hernia, so easily diagnosed, presents a mortality of only 10.8 per cent, which is about one-half of the mortality rate for the entire material. Although it represents 20 per cent of the total material, it includes only 11 per cent of the deaths. On the contrary, in femoral hernia, which is less easily diagnosed, particularly when the Richter type is present, the mortality (23.0 per cent) approximates that of the total material. These comparisons suggest that a more accurate picture of the problem of intestinal obstruction is afforded by omitting such easily diagnosed conditions as inguinal hernia from reports covering the general problem.

SUMMARY

An analysis of 406 cases of intestinal obstruction has been presented. Nine were admitted moribund.

The mortality for the entire series is 20.4 per cent, and exclusive of the moribund cases, 18.6 per cent. When cases of obstructed inguinal hernia are excluded the mortality is 22.9 per cent. Inguinal hernia represents 20 per cent of the material and 11 per cent of the deaths.

Certain well-recognized features of the disease have been re-emphasized. Some of the factors responsible for the encouraging improvement in mortality have been listed.

A distinction has been made between "reversible obstruction" and "irreversible obstruction."

The plan of treatment followed at the University of Virginia Hospital has been outlined, with emphasis on the importance of surgical intervention at the earliest possible moment in "irreversible obstruction."

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SQUAMOUS-CELL EPITHELIOMA OF THE RECTUM*

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FROM THE SECTIONS ON PROCTOLOGY AND SURGICAL PATHOLOGY, MAYO CLINIC

OPINIONS DIFFER concerning the existence as well as the origin of squamous-cell epithelioma of the human rectum. Some authorities[1-4] have expressed the belief that epidermoid carcinoma, when found in the mucosa of the rectum, is usually an extension of an anal growth and not primarily a rectal tumor. The meeting point of two different types of epithelium (modified squamous of the anus and tall columnar of the rectum) which forms the mucocutaneous junction, or the anorectal line, is thought to be the point of origin of this type of tumor when such a tumor is found in this region. The fact that squamous-cell epithelioma does occur in the rectum independent of the epithelial tissues of the anal canal is admitted by only a few investigators.[5, 6]

Although there are occasional claims that this type of lesion has been observed in the lower portion of the sigmoid and in the rectum, a search of the literature reveals that only six cases have been reported in detail. These six cases were reported by three different investigators (Raiford,[7] Cattell and Williams[8] and Keyes[9]) as squamous-cell carcinomas of the lower part of the rectum and anus.

Because of the paucity of reported cases and because the existence of this type of neoplasm in the mucosa of the rectum is doubted by some authorities, it was felt that a study of the clinical and pathologic features of these lesions was justified.

Available literature on the subject of squamous-cell epithelioma of the rectum, especially regarding squamous-cell elements in the rectum proper, is very meager. Most writers treat the subject of rectal squamous-cell cancer as part and parcel of the subject of epithelioma of the "anorectal region." In fact, we were unable to find a single article in the literature which treated exclusively the subject of rectal squamous-cell epithelioma. Recent publications offer more accurate information than those which appeared earlier.

METHODS AND MATERIAL

From 1915 through 1946, a period of 32 years, a diagnosis of rectal or anorectal squamous-cell epithelioma was made on approximately 130 patients at the Mayo Clinic. The great majority of these cases represented, in reality,

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squamous-cell epitheliomas of the anus which had or had not involved the mucosa of the rectum secondarily. It was thought that a few of these 130 cases actually represented primary rectal squamous-cell epitheliomas. The lesions in these cases were those which were "seen on rare occasions" and which occurred above and independent of the anal canal.

The clinical histories and pathologic features of this group were studied carefully in order to learn how many of the lesions were actually rectal in

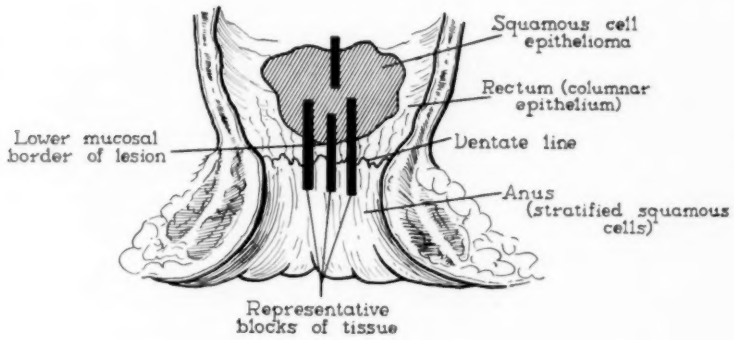

FIG. 1.—The method by which blocks of tissues were sectioned from the lowermost edge of the lesion.

origin, whether they were truly squamous-cell epitheliomas, how their presence could be accounted for, and what their clinical features were.

The pertinent data from the records of these patients included (1) clinician's résumé of the history and physical findings, (2) results of laboratory studies, (3) proctoscopic findings, (4) the surgeon's findings at operation and (5) the pathologist's report on the fresh unimbedded tissue that had not been treated with formalin.

In 14 cases surgical formalin-preserved specimens were available for this investigation. The gross study of these specimens included determination of the location of the lesion in relation to the dentate line, the approximate size of the growth, the presence or absence of ulceration, the depth of penetration of the lesion into the bowel wall and involvement of lymphatic nodes. If the anal canal appeared to be involved by the growth or if the growth appeared to originate in the anal canal final disposition of the case was not made until microscopic proof was available.

In order to prove the exact locations of the lesions, it was necessary to cut rectangular blocks of tissue through the long axis of the bowel in such a way as to include portions of the rectum, the dentate line, the anal canal and the edge of the growth which was nearest the anus. Several blocks of tissue were cut from different parts of the growth to insure that the microscopic classification of the tissue would be correct (Fig. 1).

Sections of the tissue for microscopic study were made by the frozen

section method. The hematoxylin and eosin technic was employed to stain the tissue.

On preliminary microscopic study of these 14 tumors it soon became apparent that not all of them were pure squamous-cell epitheliomas. In several instances it was necessary to determine accurately the true histologic characteristics of the lesion.

Nine tumors of the original 14 were discarded for the following reasons. Three were adeno-acanthomas (mixed adenocarcinoma and squamous-cell epithelioma). Four were atypical adenocarcinomas, of which two simulated squamous-cell epithelioma. Two were pure squamous-cell epitheliomas, but examination of the rectum showed that the squamous-cell elements did not arise from the rectal mucosa; both of these patients were women who had undergone hysterectomy, one for carcinoma of the cervix uteri and one for carcinoma of the fundus uteri.

Final evaluation yielded only five cases which could be classified as squamous-cell epithelioma of the rectum.

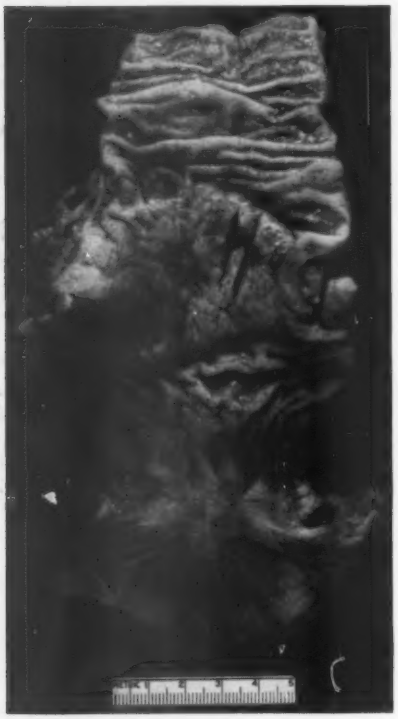

FIG. 2.—Surgical specimen in case 5 showing the gross lesion located approximately 3 cm. above the dentate line. Appearance before multiple blocks of tissue were removed.

CLINICAL OBSERVATIONS

Incidence. The infrequency of occurrence of squamous-cell carcinoma of the rectum as compared with the vast number of adenocarcinomas of the rectum reveals the rare incidence of the former. The total number of cases of squamous-cell carcinoma comprises a small fraction of one per cent of all malignant lesions of the rectum. Suffice it to say that these cases are extremely rare, almost to the point of being an academic curiosity.

Age. The youngest patient was 48 and the oldest 71 years of age. The average age was approximately 58 years, which is about the same as that of patients who have adenocarcinoma of the rectum.

Sex. There were three females and two males.

Symptoms. The cardinal symptom in this group of patients was bleeding from the rectum; it was complained of by all five patients. Two patients complained of pain, and the fact that each had type B and C lesions (Dukes) may explain the presence of pain. Constipation was mentioned by two patients.

Three to seven months was the average duration of symptoms for four of the patients, and one patient complained of trouble for a period of one year.

In comparison with adenocarcinoma, the duration of symptoms from squamous-cell carcinoma is probably a little shorter.

PATHOLOGIC FINDINGS

Location of Lesions. All five growths were located in the lowest third of the rectum but entirely above the dentate line. Four of the growths were

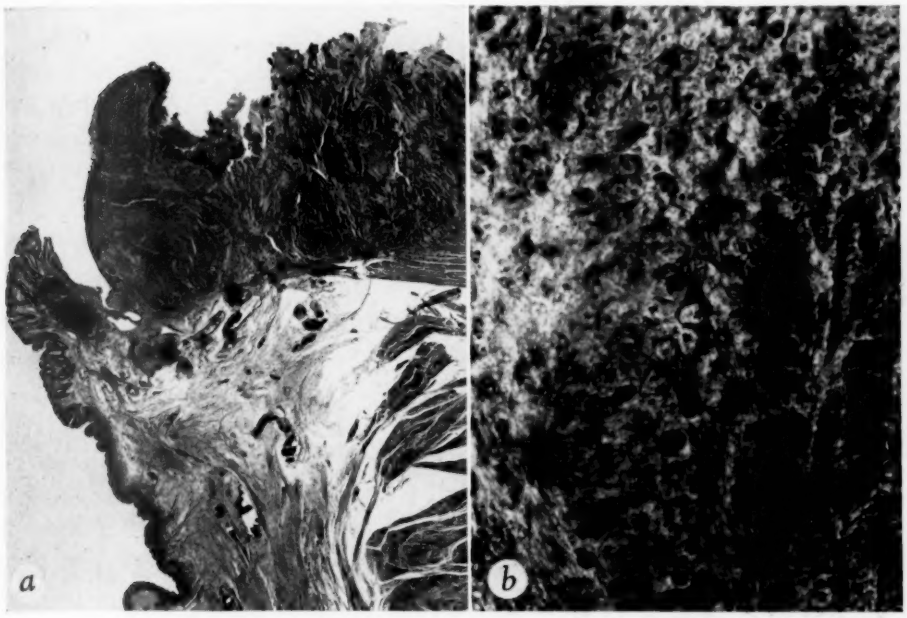

FIG. 3.—(a) A representative section from a lesion demonstrating the anal skin, the mucosa of the rectum and the squamous-cell cancer located in the rectum above the dentate line (hematoxylin and eosin x 5). (b) The cytologic features of squamous-cell epithelioma of the rectum are demonstrated (hematoxylin and eosin x 390).

located just internal to the dentate line. The lower edge of one tumor was located 3 cm. cephalad to the dentate line (Fig. 2).

Size of Lesions. The size of the tumors ranged from approximately 16 to 40 sq. cm. The thickness varied from 1 to 2 cm.

General Characteristics. Two tumors were annular and three involved about half the circumference of the bowel wall. The right and posterior walls were involved by these three lesions. Three tumors were fixed. All five were found to have some degree of ulceration. None of the tumors was polypoid.

Microscopic Picture. The cellular pattern of these lesions was similar to that observed in groups of squamous cells wherever situated. The general configuration was that of small islands of cells running in strands and cords. The fingerlike islands were separated by a sparse amount of connective tissue. These areas of cells were rectal in location. Figure 3*a* depicts the anal skin,

the dentate line, the squamous-cell lesion taking its origin from the columnar-cell mucosa of the rectum and the intervening rectal mucosa. The size of the cells varied from small to large but by far the majority were large and contained pale-staining cytoplasm. However, the cytoplasmic area was relatively reduced owing to the large size of the nucleus. The cell membranes were deep-staining and usually easy to demonstrate. In general the cells were ovoid to polygonal in shape (Fig. 3*b*).

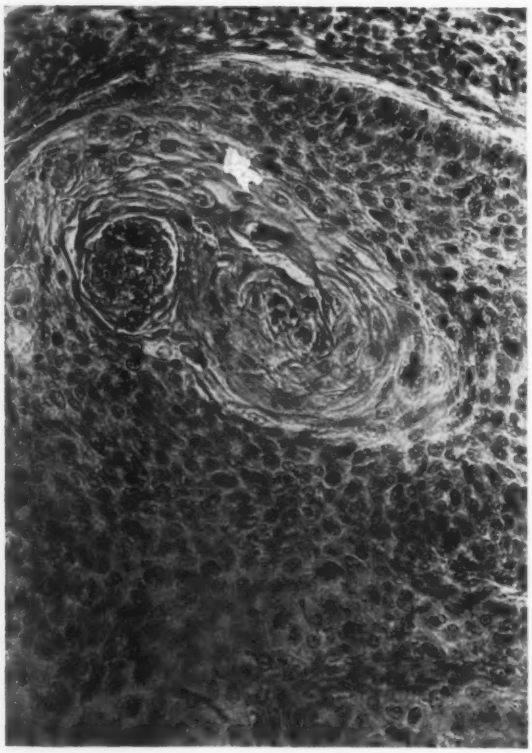

FIG. 4.—One of the rare "pearly bodies" found in the microscopic sections.

The nuclei were large and hyperchromatic and presented the appearance of a kind of granular chromatin network. Mitotic figures averaged three to four per high-powered field. Some of the mitotic figures were pathologic.

With the aid of the oil-immersion lens hyperchromatic large nucleoli could be seen. These are the "one-eyed" cells of Broders.

Grade of Lesions. The five lesions were graded according to the method of Broders. All were high-grade growths, three being Grade 4 and two being Grade 3 lesions. This is in contrast to the adenocarcinomas of the rectum which are usually low grade. We were able to demonstrate "pearly bodies" occasionally (Fig. 4). Since these bodies represent an attempt at cellular

differentiation they would not be expected to be prominent in these Grade 3 and Grade 4 lesions.

Type of Lesions. Dukes' method of determining the "bowel-wall" spread of these lesions was used. Four of the tumors were type C and one was type B.

ORIGIN OF SQUAMOUS CELLS IN THE RECTUM

It is not the purpose of this work to delve into all the possible explanations of why squamous cells are found in the rectal mucosa independent of the anorectal junction (squamous-cell lesions have been reported which were located in the mucosa of the stomach, gallbladder and large bowel other than the rectum). In our opinion Broders' explanation of "cellular metaplasia" is a most logical one. He has said: "Squamous epithelium does not originate from columnar epithelium as the columnar cell is differentiated and cannot regenerate. When a squamous cell epithelioma develops in an organ in which columnar epithelium is normally found, the gallbladder for example, the regenerative cells which under ordinary circumstances produce columnar epithelium, produce squamous cells instead. The same process takes place in an everted uterus when protective instead of columnar epithelium is needed." MacCarty[10] calls these regenerative cells the "stratum germinativum." These regenerative cells are situated in the depths of the intestinal crypts and have a pluripotential ability to produce the type of epithelium needed for a given purpose, that is, mucous cells for secreting purposes or squamous cells for protective purposes. Boyd[11] had called this phenomenon an "irritation metaplasia." MacCarty has suggested the term "anaplasia" as a more accurate one to describe this process.

METHOD OF TREATMENT

Radium is the choice of some men in the treatment of "anal" squamous-cell epitheliomas. Sweet and Cattell[12] have expressed the opinion that radical operation for squamous-cell lesions in this region is the better type of treatment.

Cattell and Williams[8] recommended abdominoperineal resection with inguinal node dissection "in every case of an operable tumor in which cure is possible and feasible." Harvey[13] recommended radical surgical treatment of the local growth for operable lesions of medium size, that is, of more than 2 cm. in diameter. He felt that extremely large lesions of this type were incurable and that lesions less than 2 cm. in diameter could be cured by wide local excision. In his experience, metastasis to inguinal nodes occurred after apparently complete removal of the rectal growth by combined abdominoperineal resection, yet he carried out inguinal nodal dissection in only a very few of his patients. In several he combined irradiation with subsequent surgical attack on residual lesions and nodes which had become involved in spite of roentgen therapy. From this and other literature on the subject it is apparent that the diagnosis of anorectal epithelioma is not often made, that when made it is frequently after considerable delay, and that with heretofore unstandardized methods of treatment, the results of therapy have been unsatisfactory.

One of our five patients was treated with radium. However, after several months there was a full-blown recurrence and the patient was then subjected to posterior resection. The remaining four patients were treated by means of more or less radical operations (Table I). Surgical treatment in our opinion would seem to be the treatment of choice for these lesions because they have the same possibilities of upward spread and lateral spread as do adenocarcinomas located in the same region. Inguinal nodal dissection was not carried out in any of the group inasmuch as no metastatic nodal involvement was clinically noted in examination of these areas. In our follow-up studies we have had no evidence that such involvement occurred subsequent to treatment. We realize, however, that in such a small series, conclusions along these lines are not justified.

TABLE I.—*Pure Squamous-cell Epithelioma of the Rectum: Clinical Features*

Case	Age and Sex	Symptoms and Duration	Grade and Type	Description of Lesion*	Treatment	Outcome
1	60 M	Constipated, red mucus by rectum; 1 mo.	4, C	4 by 4 by 1 cm.; fixed, hard	Posterior resection	Dead 6 yr. later
2	54 M	Pain and blood; 7 mo.	3, B	8 by 5 by 2 cm.; annular, ulcerative	Posterior resection	Dead?
3	48 F	Constipation and blood; 4 mo.	4, C	4 by 4 by 1 cm.; large posterior wall	Radium and posterior resection	Dead 2 yr. later
4	56 F	Pain and blood; 3 mo.	4, C	4 by 4 cm.; posterior wall, fixed	Combined abdominoperineal resection	Alive 2 yr. later
5	71 F	Blood by rectum; 5 mo.	3, C	5 by 6 by 2 cm.; annular, fixed	Posterior resection	Alive. Operated on July 20, 1947

* All lesions on the lower part of the rectum.

PROGNOSIS

One patient survived for six years after operation and then died of recurrence. One patient died two years after operation but we were unable to ascertain the cause of death. The third patient failed to answer a letter of inquiry. The fourth was alive two years after operation. The fifth patient was alive and well three months postoperatively, but her lesion was a Grade 3 (Broders), type C (Dukes) tumor and her outlook was not good.

CONCLUSIONS

1. Squamous-cell epithelioma occurs rarely in the rectum and independently of the dentate line of the anus. The five cases herein reported support this belief.

2. Clinically, the symptomatology of these lesions is similar to that of adenocarcinoma of the rectum.

3. Pathologically, all five lesions were high grade, that is, Grades 3 and 4 (Broders), whereas adenocarcinomas are usually low grade, that is, Grades 1 and 2.

4. Adeno-acanthomas, adenocarcinomas that imitate squamous-cell epithelioma, and metastatic lesions from the cervix uteri may be mistaken for primary squamous-cell epitheliomas of the type under discussion.

5. The treatment of choice seems to be radical operation.

6. The prognosis is very poor.

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FAILURE OF DICUMAROL* TO INTERFERE WITH ADHESIVE LOCALIZATION OF INTRA-ABDOMINAL INFECTIONS†

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THE USE OF ANTICOAGULANTS to prevent the formation of intra-abdominal adhesions has been enthusiastically reported in the literature on numerous occasions during the past few years of Lehman and Boys.[1-5] They have employed heparin intraperitoneally following enterolysis and have noted that the extent of reformation of adhesions in dogs so treated was only one-seventh to one-eighth that observed in the control series.

Dicumarol is another anticoagulant that has found a wide field of usefulness during recent years, especially in the treatment of thrombophlebitis and the prevention of postoperative thrombosis and embolism. Many writers have expressed fear in the use of Dicumarol in the presence of intraperitoneal infection, the argument being that anticoagulants encourage a spreading peritonitis by preventing the natural walling off tendency of the peritoneum and omentum in the presence of infection. The ability of an intra-abdominal infection to wall itself off into a localized abscess is a function of adhesive formation, and nothing should be done that will in any way hinder this walling off tendency. The purpose of this study is twofold: (1) to determine the effect of Dicumarol on the reformation of adhesions following enterolysis and (2) to determine the effects of Dicumarol in the presence of intraperitoneal infection, especially as related to any interference of the drug with the walling off tendency of experimentally produced appendicitis with perforation.

EFFECT OF DICUMAROL ON ADHESIVE FORMATION

Technic Employed. Adhesions were produced as follows: Under intravenous nembutal anesthesia (15 mg. nembutal per lb. of body weight) laparotomy was performed through a midline incision and the terminal two feet of small bowel was traumatized with dry gauze with sufficient force to produce a roughening and capillary oozing from the serosa. Healthy dogs were used exclusively. The traumatized bowel was then painted with a 7 per cent solution of tincture of iodine and the abdomen closed in layers, using 000 silk. Two to three weeks later laparotomy was repeated and the extent of adhesive formation carefully estimated, the adhesions divided by sharp dissection with careful attention to hemostasis, and the abdomen closed. Dicumarol by mouth was begun two to three days prior to the second laparotomy, prothrombin

* Dicumarol is a registered collective trademark owned by the Wisconsin Alumni Research Foundation, which controls the use thereof.

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time determinations showing a 2:1 to 4:1 ratio at the time adhesions were divided, and was given daily postoperatively in sufficient doses to maintain prothrombin time ratios ranging from 2:1 to 5:1. Dicumarol therapy was continued up to seven days postoperatively and laparotomy again repeated at varying intervals and the extent of reformation of adhesions estimated.

METHOD OF DETERMINING PROTHROMBIN TIME

The following method described by Quick was used to determine the prothrombin times: Blood was drawn from the anterior cubital vein each morning prior to the administration of the Dicumarol. Freshly prepared thromboplastin (0.1 cc.) was placed on one side of a hanging-drop type of

TABLE I.

Dog No.	Prothrombin Time Ratio at Time of Enterolysis	Av. Daily Dose of Dicumarol (mgs.)	Average Prothrombin Time Ratio	No. Days Dicumarol Given Following Enterolysis	Extent of Reformation of Adhesions	Comments
40	2.7:1	25	2.7:1	5	Same	
52	.2:1	35	3.6:1	4	Same	Dog expired 5th p. o. day from massive bleeding into bowel
53	1.8:1	40	4.1:1	5	Same	Dog expired 5th p. o. day from massive intra.-abd. bleeding
57	3.4:1	20	4.2:1	1	Fibrinous adhesions present	Dog expired from peritonitis 2nd p.o. day
59	2.1:1	25	2.3:1	5	More extensive	
61	3.6:1	26	6.6:1	1	Fibrous adhesions present	Expired 2nd p.o. day abd. filled with blood
60	5.4:1	23	5.3:1	6	More extensive	
62	2.6:1	24	3.1:1	7	More extensive	

glass slide and 0.1 cc. of calcium chloride on the other side, not permitting the two drops to mix. Then, 0.1 cc. of blood from the anterior cubital vein was placed between the two drops and the three drops immediately mixed and the time measured with a stop watch for fibrin formation to occur. Normal healthy dogs were used as controls. Eight dogs were treated with Dicumarol in the manner described above. Table I shows the essential data of this series. Five dogs served as controls, receiving no Dicumarol prior to and following enterolysis.

DISCUSSION

This experiment proves definitely that Dicumarol is inffectual in preventing the reformation of divided adhesions. Prothrombin time ratios ranging from 2:1 to as high as 12:1, with an overall average of 3.8:1 were present at the time of division of the adhesions and maintained for periods up to

seven days postoperatively. In all these Dicumarol treated dogs the extent of adhesion reformation was as great as or usually greater than the degree of adhesions present prior to treatment with the drug. An exact quantitative study, as suggested by Lehman and Boys, of the amount of adhesive formation was not possible in as much as the usual finding at operation was a

TABLE II.—*Dicumarol.*

Dog No.	Weight	Penicillin and Sulfadiazine	Daily Dose of Dicumarol (Mg.)	Prothrombin Time Ratio (Average)	No. of Days P.O. Dicumarol Given	Course of Disease	Autopsy Findings
76	15	No	20 mg.	4.6:1	3	Expired 5th p.o. day	Autopsy revealed a partially walled-off appendiceal abscess with local peritonitis.
77	15	No	25 mg.	2.5:1	11	Sacrificed 20 days p.o.	Well walled-off appendiceal abscess, 1x2 cm. No evidence of generalized peritonitis.
78	16	No	25 mg.	3.2:1	0	Expired 2nd p.o. day	Localized appendiceal abscess containing 100 cc. pus. Dense adhesions of loops of small bowel.
79	13	No	18 mg.	3:1	6	Expired 6th p.o. day	Partly walled-off appendiceal abscess. Free pus in peritoneal cavity.
82	16	No	25 mg.	1.9:1	2	Expired 2nd p.o. day	Appendix black and had sloughed. Small amount of foul-smelling fluid in peritoneal cavity.
66	16	Yes	21 mg.	2.5:1	12	Sacrificed 15 days p.o.	Well walled-off appendiceal abscess. Dense adhesions between abscess wall and parietal peritoneum.
67	21	Yes	35 mg.	2.1:1	13	Sacrificed 18 days p.o.	Dense adhesive mass over ileocecal region. When these adhesions were broken down a small abscess was encountered.
68	17	Yes	20 mg.	3.1:1	13	Sacrificed 15 days p.o.	Well walled-off appendiceal abscess containing about 2 cc. pus. Peritoneal cavity clean.
70	23	Yes	35 mg.	2.9:1	10	Sacrificed 13 days p.o.	Well walled-off appendiceal abscess containing about 150 cc. pus. Peritoneal cavity clean.
72	20	Yes	25 mg.	1.4:1	3	Expired 3 days p.o.	200 cc. sanguineous-purulent fluid in peritoneal cavity. Appendix sloughed and partially walled-off by adhesions.
75	19	Yes	30 mg.	2.9:1	8	Expired 9 days p.o.	About 200 cc. purulent material in peritoneal cavity. Appendix necrotic and partially walled-off by adhesions.
85	21	Yes	42 mg.	3.1:1	9	Sacrificed 11 days p.o.	Well walled-off appendiceal abscess containing about 150 cc. pus. Peritoneal cavity clean.
84	23	Yes	40 mg.	2.6:1	3	Expired 4 days p.o.	Walled-off abscess containing 100 cc. purulent fluid. About 300 cc. purulent fluid in preperitoneal cavity.
87	15	Yes	24 mg.	2.5:1	6	Sacrificed 8 days p.o.	Well walled-off appendiceal abscess size of walnut. Peritoneal cavity clean.
88	12	Yes	21 mg.	3.4:1	6	Sacrificed 8 days p.o.	Well walled-off appendiceal abscess containing about 3 cc. pus. General peritoneal cavity clean.
80	12	Yes	23 mg.	4 :1	6	Expired 7 days p.o.	Well walled-off appendiceal abscess. 150 cc. serosanguineous fluid in peritoneal cavity. Pneumonia involving entire right lung.

coiling up of the traumatized bowel in an adherent mass with omentum densely adherent to this mass and to the parietal peritoneum. But it was readily apparent that the extent of adhesion formation was just as great in the dogs treated with Dicumarol as in those not receiving the drug. The

danger of hemorrhage was well demonstrated in this small series of Dicumarol treated dogs. Three of the eight dogs so treated expired from bleeding.

DICUMAROL IN THE PRESENCE OF INTRA-ABDOMINAL INFECTION

Having shown the ineffectiveness of Dicumarol on intra-abdominal adhesive formation an experimental study was next carried out to determine the relationship between the use of Dicumarol and the ability of the contaminated peritoneum to wall off the infection. The report of Kay and Lockwood[6] on

TABLE III.—*Dicumarol.*

Dog No.	Weight	Penicillin and Sulfadiazine	Course of Disease	Autopsy Findings
23	23	No	Expired 6th p.o. day	Generalized peritonitis. About 300 cc. purulent fluid in abdominal cavity. Appendix completely sloughed and unidentifiable.
86	8	No	Expired 10th p.o. day	Generalized peritonitis with no walled-off tendency present.
56	12	No	Expired 8th p.o. day	Partially walled-off appendiceal abscess size of hen's egg with local peritonitis.
89	20	No	Expired 8th p.o. day	Gangrenous perforated appendix with generalized peritonitis.
90	15	No	Expired 2nd p.o. day	Appendix sloughed and entire pelvic cavity filled with fecal pus.
63	12	Yes	Sacrificed 19 days p.o.	Well walled-off abscess about 1 cm. in diameter. No evidence of peritonitis.
65	13	Yes	Sacrificed 16 days p.o.	Well walled-off appendiceal abscess size of walnut. General peritoneal cavity clean.
69	16	Yes	Expired 2nd p.o. day	Generalized peritonitis. Appendix sloughed. Open hole in cecum leaking fecal material.
71	17	Yes	Sacrificed 17th p.o. day	Well walled-off appendiceal abscess size of lemon. General peritoneal cavity clean.
73	11	Yes	Sacrificed 16th p.o. day	Walled-off appendiceal abscess size of walnut. No evidence of generalized peritonitis.
74	18	Yes	Sacrificed 17th p.o. day	Walled-off abscess size of grape. No evidence of generalized peritonitis.
81	12	Yes	Expired 2nd p.o. day	Perforated gangrenous appendix with generalized peritonitis. No adhesive formation about appendix.
91	18	Yes	Expired 2nd p.o. day	Generalized peritonitis. Appendix gangrenous and had perforated at tip. No walling off tendency.
59	13	Yes	Sacrificed 30 days p.o.	General peritoneal cavity free of infection. Dense adhesions about ileocecal region. Small necrotic core found when adhesions were broken down.
92	16	Yes	Expired 4th p.o. day	Generalized peritonitis.
93	13	Yes	Expired 2nd p.o. day	Gangrenous appendix with collection of free pus in ileocecal region.

the prognostic significance of the prothrombin time in experimental peritonitis is interesting. They produced gangrenous appendicitis with perforation and peritonitis in a series of 22 dogs. Ten of their series exhibited a normal prothrombin time with a mortality of 10 per cent and 12 exhibited increased prothrombin times varying from 1.5; 1 to 2:1 ratios with a mortality of 83 per cent. No mention is made in their paper as to any correlation between prothrombin time and tendency for the peritoneal infection to wall itself off.

TECHNIC EMPLOYED

Gangrenous appendicitis with perforation and abscess formation was produced as follows: under intravenous nembutal anesthesia all blood supply to the appendix was carefully tied off and the base of the appendix ligated with heavy cord. In all instances the appendix became gangrenous, perforated, and resulted in either local or generalized peritonitis. The local peritonitis either walled off to abscess formation or went on to spreading peritonitis and death. Half of these dogs were treated with Dicumarol and half were used as controls. In the early part of the experiment no chemotherapy or penicillin was employed. The mortality rate was very high. Thereafter, supportive therapy with intravenous fluids, sulfadiazine, and penicillin resulted in a substantial decrease in the mortality rate. A total of 32 dogs was employed in this experiment.

Table II summarizes the essential data in the Dicumarol treated series and Table III in the series not receiving Dicumarol.

DISCUSSION

A study of Tables II and III shows that Dicumarol has no untoward effect on the ability of the contaminated peritoneum to localize and wall off the infection. Even in the dogs that expired in the Dicumarol-treated series varying degrees of walling off of the gangrenous appendix were present at autopsy. The mortality rate (Table IV) was actually less in the Dicumarol treated series. Goth[8] has observed the growth inhibitory effect of Dicumarol in 14 different bacterial species and found it to possess marked anti-bacterial properties.

TABLE IV.

Type of Therapy	No. of Dogs	Mortality Rate
Dicumarol	5	80%
Dicumarol, penicillin and sulfadiazine	11	36%
No drugs	5	100%
Penicillin and sulfadiazine	11	45%

The dosage of penicillin used was 100,000 to 200,000 units per day in divided doses and 4 Gm. of sulfadiazine were added to the I. V. fluids for a period of four to five days postoperatively. In 11 of the 16 dogs receiving Dicumarol, the drug was started on the day following surgery. In dogs 85, 84, 87, 88, and 80 (Table II) Dicumarol was started preoperatively with a prothrombin time ratio of an average of 3.3 at the time of the surgical procedure. This did not prove to be a factor of any importance in determining whether a local or a generalized peritonitis would result.

SUMMARY

1. Dicumarol orally has no effect on the formation of intra-abdominal adhesions.

2. The walling off of an intra-abdominal infection is in no way interfered with by the oral administration of Dicumarol.

3. The mortality in a series of dogs with gangrenous appendicitis going on to perforation and formation of either local or generalized peritonitis was not increased by the use of Dicumarol.

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A TECHNIC FOR THE VISUALIZATION OF EXPERIMENTAL PEPTIC ULCER FORMATION IN THE DOG*

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IN 1923 MANN AND WILLIAMSON[1] described an operative procedure for producing peptic ulcer consistently in dogs by internal duodenal drainage and gastrojejunal anastomosis. The animal preparation described by them has, doubtless, been used more often than any other for studies of peptic ulceration. The method, nevertheless, contains inherent disadvantages, among which are: (1) the difficulty of maintaining the animal in a good nutritive state; (2) the unfeasibility of determining the time of appearance and course of the ulcer accurately except by repeated laparotomies; and (3) the fact that the lesion differs from human stoma ulcer so that, for example, the preparation is unsuitable for studies of vagotomy because, as Dragstedt[2] observed, "One obvious difference between the two lesions depends upon the fact that in experimental ulcer the duodenal secretions are deviated completely into the lower intestine while in most stoma ulcers in man these secretions pass over the ulcer area."

Because of these limitations of the Mann-Williamson operation it was decided to attempt an improved technic which would provide the obvious advantages of easy visualization of the lesion and the maintenance of a good nutritive state.

Matthews[3] described in 1931 the production of peptic ulceration in dogs by the method of anastomosing a Pavlov pouch to an isolated segment of ileum which, in turn, was drained into the ileum proper. In his animals, ulceration always formed within a short distance of the anastomosis between the gastric pouch and the segment of ileum. Thomas[4] described in 1941 a modification of the Pavlov pouch in which there was minimal disruption of the vagal innervation. In the work here described both of these technics, in modified form, were combined in a single preparation.

METHODS

Three adult, female, mongrel dogs, of about 20 Kg. body weight, were anesthetized with intravenous pentobarbital. A high midline incision was made, extending about eight to ten centimeters downward from the xiphoid process. Through this incision the stomach was delivered to the fullest extent possible by gentle manipulation. The extent and location of the pouch was outlined, using two Allis clamps as markers (Fig. 1a). Midway between the marker clamps and close to the greater curvature a 1.5 cm. incision was made perpen-

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dicular to the greater curvature and extending into the gastric lumen. Through this incision, two Allis clamps were introduced and the stomach wall grasped, from within, at the sites of the external marker clamps (Fig. 1a). The outside marker clamps were then removed. With the inner clamps the gastric wall was everted through the small incision to form a large rosette (Fig. 1b). A ribbon of mucosa, about 0.5 cm. wide, was excised from the crest of the rosette, with curved scissors. This exposed an elliptical band of submucosa, the poles of which lay at the Allis clamps (Fig. 1b). Bleeding was rather free, even after the larger vessels were ligated. Hemorrhage can be reduced and a neater field obtained by applying long Doyen clamps but, in any case, the bleeding was more inconvenient than serious and clamps were not used in the cases here

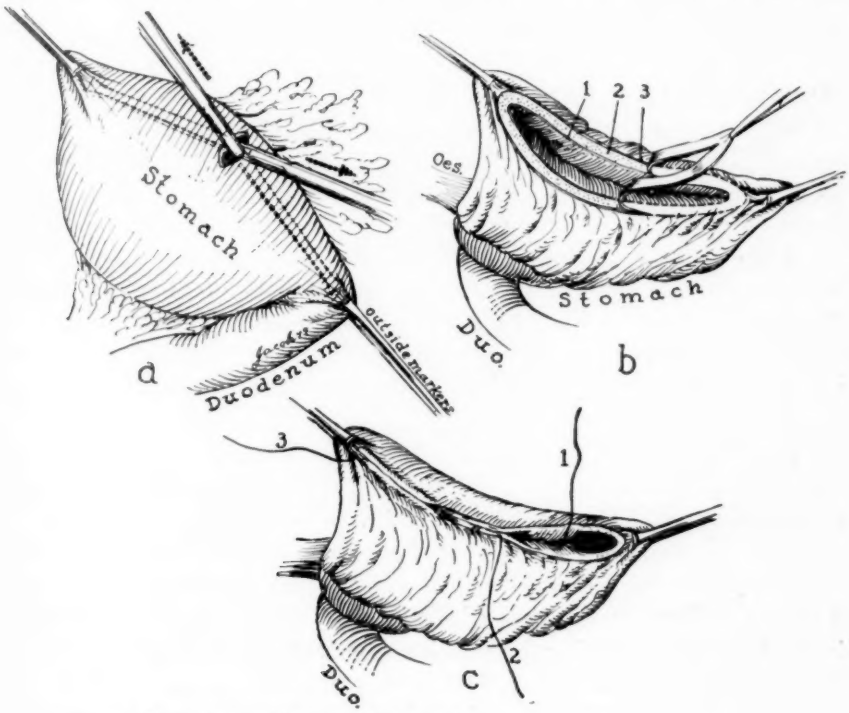

FIG. 1.—Steps in making gastric pouch.

a—selecting site and preparing to evert stomach.
b—excision of mucosal ribbon
c—creation of septum separating pouch from gastric lumen proper.

reported. The inner-most mucosal layer, of the gastric lumen proper, was brought together with a Connell suture so that raw surfaces were apposed (Fig. 1c, layer 1), special care being taken at the corners. The submucosa was next brought together with a running mattress suture (Fig. 1c, layer 2), reinforced by a few interrupted mattress stitches, paying special attention again to the corners. This submucosal suture line, consisting of shallow bites involving little or none of the muscularis, constitutes the "bulwark," or

strongest layer, of the septum which divides the pouch from the stomach proper. The outer mucosal layer, forming the dome of the pouch, was closed with a running mattress suture (Fig. 1c, layer 3) which again apposed raw surfaces. After completion of this three layered septum, the everted pouch was replaced (Fig. 2a).

A loop of intestine was next selected, and to this the pouch was anastomosed by the usual two suture line method, utilizing the original incision made in the stomach (Fig 2a). The loop of intestine selected for this purpose must be at

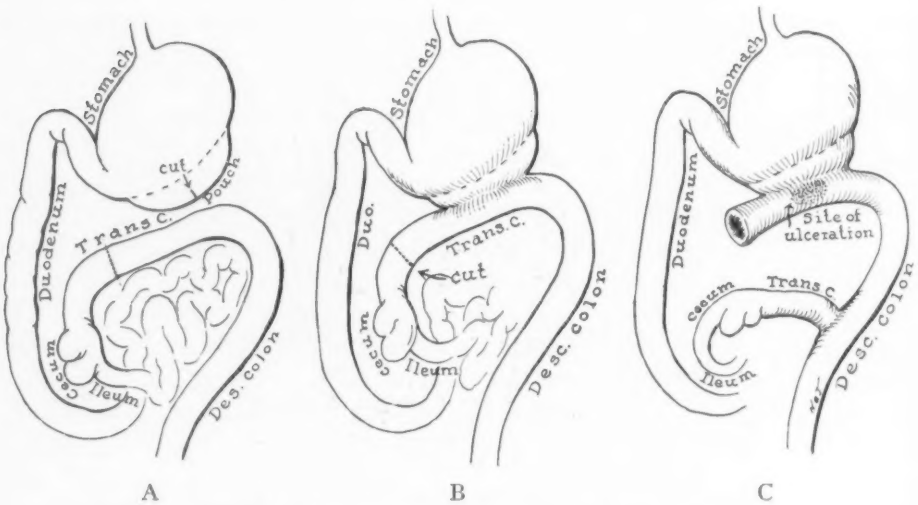

FIG. 2.—(A) Selection of intestinal loop. (B) Intestinal loop anastomosed to gastric pouch, site of division of selected portion of intestine. (C) End-to-side anastomosis re-establishing intestinal continuity.

least 25 cm. below the ligament of Treitz, but can be as far below this as desired. After anastomosing the pouch to the loop selected, the intestine was divided between clamps about 16 to 18 cm. oral to the pouch-intestine anastomosis. The aboral side of this division was brought out of the abdomen by means of a stab wound through one of the rectus muscles, and placed so that the intestinal segment between stab wound and pouch was not kinked or redundant (Fig. 2b). The oral end of the divided intestine was then anastomosed, by the oblique end-to-side method, to the intestine distal to the pouch-intestine anastomosis (Fig. 2c), thus re-establishing intestinal continuity.

In dog No. 1, the pouch was anastomosed to the transverse colon, and the hepatic flexure brought over and anastomosed to the descending colon about 15 cm. below the pouch-colon anastomosis (Fig. 2c). In dog No. 2, the pouch was anastomosed to the mid-ileum, and the oral divided end anastomosed to the terminal ileum about 15 cm. below the pouch-ileum anastomosis. In dog No. 3 the pouch was anastomosed to the mid-jejunum and the oral end of the

divided jejunum anastomosed about 40 cm. below the pouch-jejunum anastomosis.

The abdomen was closed in layers in the usual fashion and a "button" made of the intestine protruding from the stab wound, using interrupted sutures to the fascia and skin. Number 60 cotton was used for the submucosal suture line of the pouch and for all sero-serosal suture lines of the anastomoses. For all other suture lines, either number 60 cotton or fine chromic gut (No. 000 or No. 0000) was used, apparently with equal success and similar results.

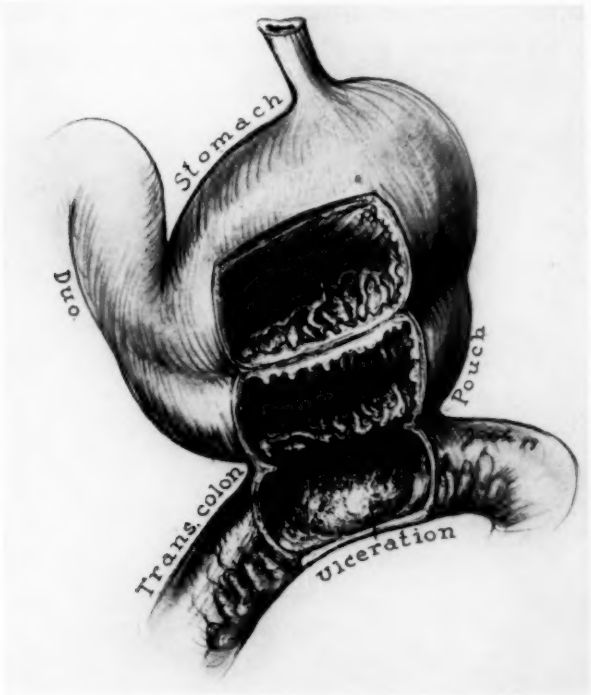

FIG. 3.—Dog 1. Gross postmortem specimen. (Artist's drawing.)

During the first two days after operation, the dogs were maintained exclusively by intravenous feedings which included glucose, saline and protein hydrolysate. On the third day water was permitted; on the fifth day soft food, and by the seventh day, a full diet of hospital kitchen scraps was given. Procaine-penicillin-G, 200,000 units, was administered intramuscularly daily for four days after operation.

By means of the procedure described above, a gastric pouch with intact innervation is caused to drain into a selected loop of intestine. The area of this drainage can be completely removed from contact with normal intestinal contents simply by placing it a sufficient distance from the end-to-side anastomosis, which diverts the chyme delivered from the upper intestine. The segment of

intestine opening out of the abdomen is a Maydl type enterostomy, and affords access to the ulcer area with the aid of an enteroscope. Normal peristaltic movements inward prevent significant leakage of gastric or intestinal juice to the outside.

On the tenth postoperative day and thereafter, detailed visual examination of the pouch-intestine anastomosis area was carried out with an infant size proctoscope having a diameter of 1.2 cm.

In the first dog, large intestine was used to permit easier examination until experience should be gained with the method, but subsequent examination of the small intestine proved to be no more difficult. In one animal, spasm of the intestine was a hindrance, but this was overcome with a single 50 mg. oral dose of trasentin 30 minutes before examination.

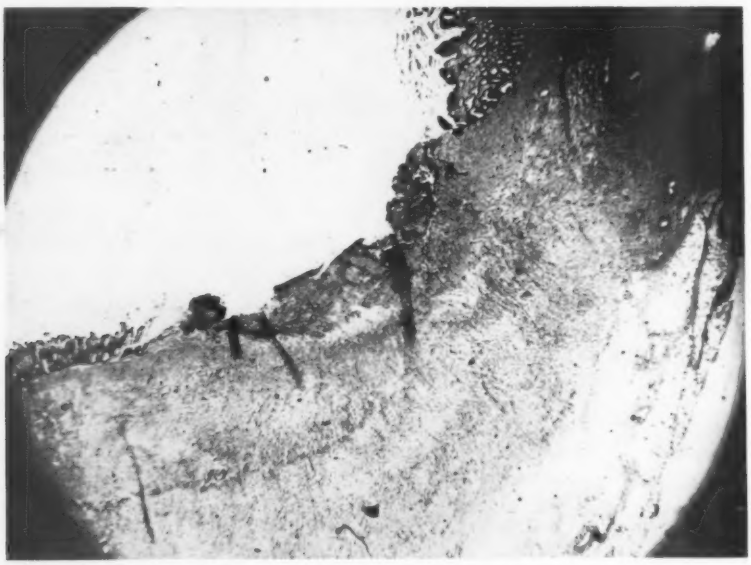

FIG. 4.—Dog 2. Photomicrograph of section through ulcer area under low magnification. This shows ulcer crater and marked fibrosis replacing muscularis.

RESULTS

There were no operative deaths and no undue postoperative morbidity. There were no infections and in no case was there any communication between the gastric pouch and the stomach proper. All suture lines remained intact.

The dogs remained in an excellent state of nutrition with no weight loss until ulcer pathology was definite, at which time irregular vomiting made its appearance, with resultant weight loss evident in two of the three animals.

All animals developed peptic ulceration in the area of the intestine immediately adjacent to or directly opposite the pouch-intestine anastomosis. In dog No. 1 (colon) an ulcer was first noted on the twenty-ninth postoperative day,

and death due to massive hemorrhage from the ulcer occurred on the seventy-first day. In dog No. 2 (ileum) an ulcer was first noted on the twenty-fourth postoperative day. Dog. No. 2 was sacrificed on the sixty-third postoperative day. At this time, the animal was in relatively good condition, though vomiting had been in evidence for several weeks with resultant weight loss. In dog No. 3 (jejunum) an ulcer was first noted on the tenth postoperative day. This animal was sacrificed on the fourteenth postoperative day. The animal was in excellent condition despite the onset of irregular vomiting several days before.

PATHOLOGY

All lesions occurred in the area of the intestine immediately adjacent to or directly opposite from the stoma of the pouch-intestine anastomosis. Dog No. 1 showed a denuded area about 3 cm. in diameter, with marked thickening of the colonic wall (Fig. 3). Microscopic section showed a clean demarcation between normal mucosa and the ulcerated area. A very marked fibrous tissue proliferation replaced the muscularis, with conspicuous thickening of the intestinal wall. The attached photomicrograph (Fig. 4) is of the lesion found in dog No. 2. The pathologic changes in dogs No. 1 and No. 2 were quite similar. The findings in dog No. 3 resembled those in dogs No. 1 and No. 2 but, since this animal was sacrificed early, the proliferative reaction and resultant thickening of the intestinal wall were not so pronounced.

All of these lesions were very similar to, if not identical with, the subacute and chronic forms of peptic ulcer seen in the human.

DISCUSSION

A surgical method is described whereby peptic ulceration was produced at three levels in the intestinal tract of the dog. The time of appearance of the ulcer and the course of the lesion were easily visualized in detail.

Several factors must be considered in the application of this procedure, namely:

1. The size and location of the gastric pouch affects the volume and nature of the gastric juice which drains into the intestinal segment.

2. The level of intestine used for the pouch-intestine anastomosis is a significant factor, since it is well known that resistance to ulceration decreases as one progresses aborally along the intestinal tract.

3. The size of the pouch-intestine stoma may be significant in determining the speed of appearance and progress of ulceration.

4. The distance from the pouch-intestine anastomosis to the entero-anastomosis would appear to be most significant up to a critical distance of about 25 cm. Pearse, Radakovich, and Cogbill[5] (1949) have shown that reflux up the small intestine, in the antiperistaltic direction is possible to the extent of about 25 cm. They do not specify whether this varies with the level of intestine under consideration. The amount of such reflux determines the volume of acid-binding intestinal contents which passes over the ulcer area. In dog No. 3, for

example, this distance was about 40 cm., which may explain the rapidity of formation of the ulcer in the animal.

The modified Pavlov gastric pouch was selected for this series because its innervation probably remains intact. Any other type of pouch, however, may be utilized for this purpose, including the entire isolated stomach, depending upon the purpose for which it is intended.

This preparation appears to offer the following advantages over other types: (1) it permits accurate determination of the first appearance and course of the lesion; (2) it is possible to maintain good nutrition in the animal; (3) the usual conditions of human stoma ulcer can be closely approximated. If the distance from the pouch-intestine anastomosis to the entero-anastomosis is made very short, then by copious reflux, an essentially normal amount of intestinal content will pass over the ulcer area; (4) it perhaps offers a better controlled preparation in that the causative agent is uncontaminated gastric juice from a pre-selected area of the stomach.

A consideration of the mortality and morbidity data on the three animals here presented indicates the feasibility of the method and that the operation is not unduly difficult. It is easily managed and completely satisfactory for the purpose for which it is intended. It is suggested that anyone interested in peptic ulcer research may find it a useful preparation.

The authors are grateful to Dr. R. Blandeau of the Department of Anatomy for the photomicrography and to Mr. N. Jacobs of the Department of Medical Art for the illustrations incorporated in this work.

SUMMARY

1. The shortcomings of earlier methods for the production of experimental peptic ulcer in dogs are discussed.

2. A new technic is described which possesses several advantages, the greatest of which is access to the ulcer bearing area for visual examination.

3. Ulcers were produced and visualized in the colon, ileum and jejunum.

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PROXIMAL FEMORAL VENOGRAPHY

A PRELIMINARY REPORT*

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A KNOWLEDGE of the physiologic and anatomic status of the deep and superficial venous systems of the lower extremity is of paramount importance before rational therapy can be directed to the correction of existing defects. Venography in the past has not given entirely satisfactory results. The selection of cases for femoral vein ligation has been based largely upon physical findings correlated with a history of past thrombophlebitis. We have become interested in developing a diagnostic roentgenologic method that will give additional information concerning the deep venous system of the lower extremity. To be of more value than previously reported methods it must permit visualization of the deep venous system under conditions as nearly approaching normal as possible. To achieve this end the patient should be in the upright position and the extremity should be unencumbered by tourniquets or bandages. With these conditions in mind we have developed a method of venography by placing a catheter in the proximal stump of the saphenous vein or in one of the saphenous tributaries in the upper thigh just distal to the saphenous bulb. Injection of 35 per cent diodrast followed by serial roentgen ray films is then carried out with the patient in the erect position. Demonstration of normal and abnormal deep venous systems by this method has been so satisfactory to us that we feel we have been able to place subsequent surgical therapy on a more rational basis.

It is beyond the scope of this paper to review the voluminous literature on venography that has accumulated since Berberich and Hirsch[1] injected radiopaque material directly into the lumen of a vein in 1923. Direct venography for the purpose of demonstrating obstructive lesions in veins was first done by Barker and Camp[2] and reported in 1936. Since that time numerous excellent reports of technic of venography and interpretation of results have appeared and those interested should review the reports of Dos Santos,[3] Bauer,[4, 5] Baker[6] and others.

Luke[7] reported in 1943 a technic of retrograde venography by direct needle injection of the femoral vein below the crease of the groin and concluded that the method was of value when a clear filling of the femoral vein was desired

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FIG. 1.—(Case 1) I. Y., 62-year-old male with history of varicose veins of many years duration; no history of thrombophlebitis. Examination revealed moderate superficial varicosities with no edema, pigmentation or ulceration. Venograms by injection of proximal saphenous stump performed. Note the limitation of retrograde flow, the easily visualized valve stations, the absence of dilated communicating veins, and the rapid clearing of the contrast medium. This is considered to represent a normal venogram by the described technic.

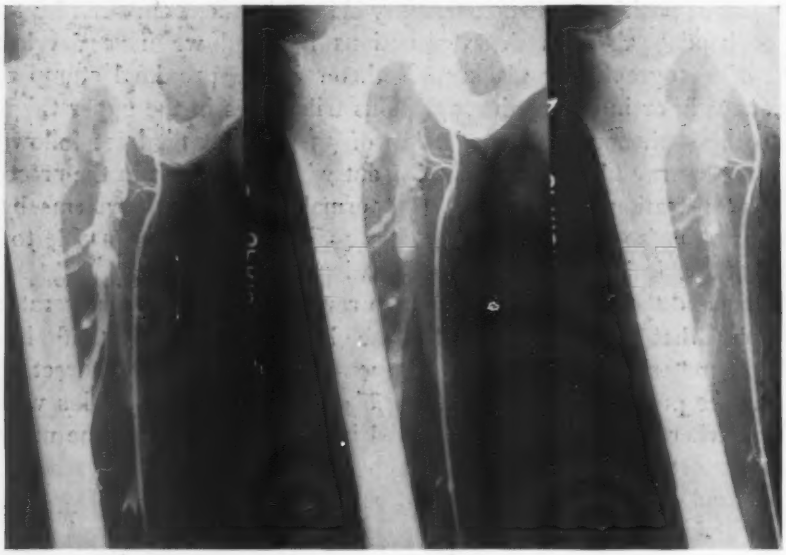

FIG. 2.—(Case 2) J. F., 64-year-old white male with history of right femoral thrombophlebitis following an abdominal operation 20 years previously. Examination revealed large tortuous superficial varicose veins, lower leg edema, pigmentation and dermatitis. Proximal femoral venography was performed by injection through the proximal stump of the saphenous vein. There is marked dilatation of the superficial femoral vein with retrograde fall of the contrast medium through both the superficial and deep femoral veins and failure to clear at the end of four minutes. The valve stations are easily identifiable but none are competent enough to prevent the retrograde flow.

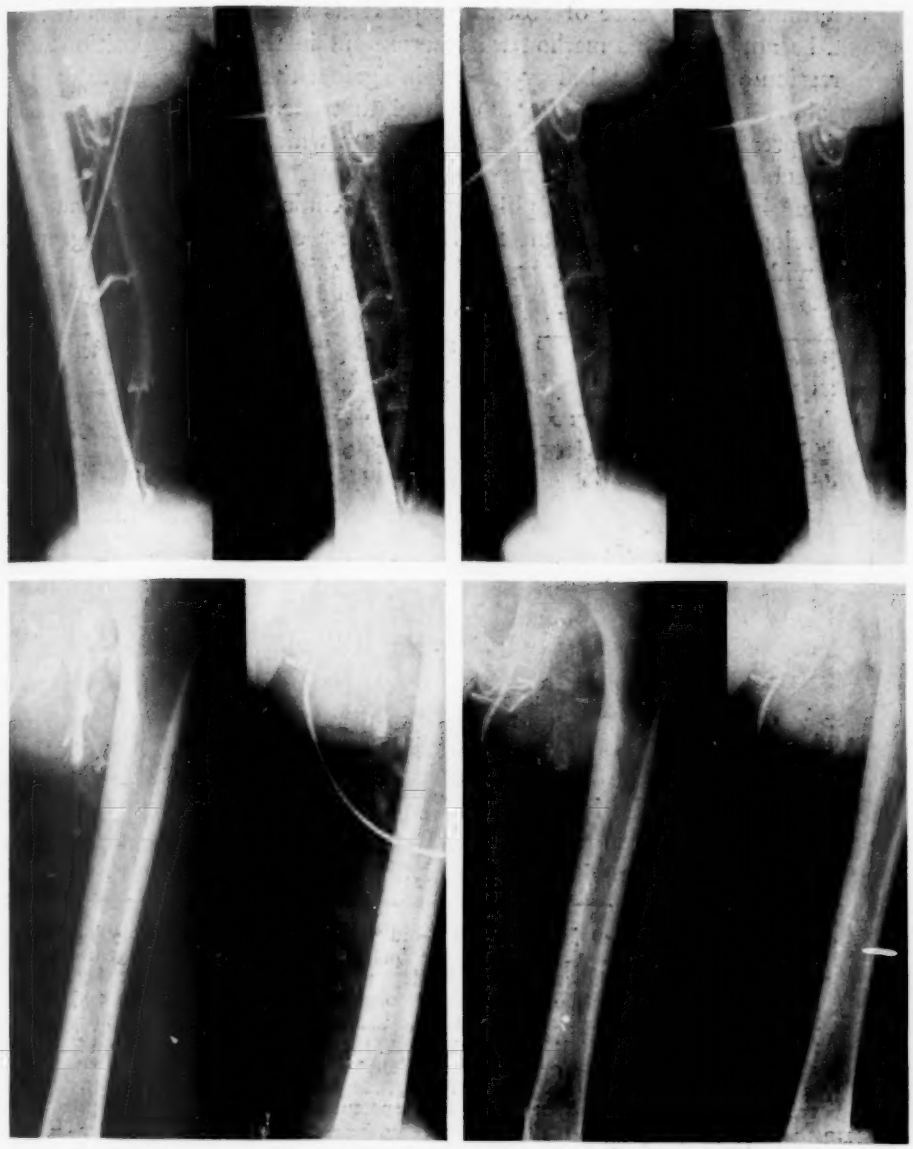

FIG. 3.—(Case 3) A. S., 67-year-old white male with the complaint of bilateral varicosities of 35 years duration. History suggestive of right femoral thrombophlebitis 40 years previously following an infection resulting from burns to both legs. Examination revealed large superficial varicosities with cellulitis, ulceration and old burn scars. The right leg was more involved than left. Right proximal femoral venogram done on April 2, 1949. Note the retrograde flow throughout the deep circulation, the dilated communicating veins and the persistence of the contrast medium in the lumen of the deep veins at one, two and four minutes after injection (upper row).

Left femoral venogram (lower row) shows the absence of retrograde fall, no outline of communicating vessels between the superficial and deep circulation, and rapid clearing of the contrast medium.

to determine the presence or absence of previous silent femoral phlebitis. It should be noted that his method was carried out in the supine position with compression of the femoral artery and vein against the superior ramus of the pubis above the site of injection. We feel that such a procedure produces artificial pressure changes in the femoral system which obscure the true state of the venous circulation.

Bauer[8] in 1948 reported a phlebographic technic essentially the same as that developed by us and in an excellent review correlates his venographic studies with operative findings and microscopic sections.

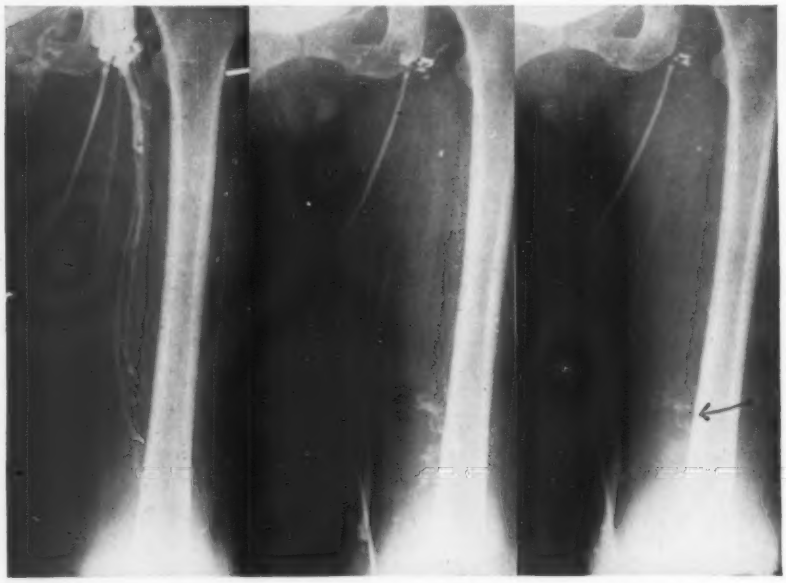

FIG. 4.—(Case 4) R. P., 44-year-old white female with history of left leg thrombophlebitis following the birth of her last child 20 years ago. Examination showed the right leg to be normal. The left leg showed edema, pigmentation and ulceration. Venograms were carried out by the proximal femoral injection technic in March, 1949. A retrograde fall through a double lumened deep vein without evidence of normal valve stations was visualized. There was failure to clear the contrast medium in four minutes (arrow). Note the retrograde fall through the saphenous vein to the level of the knee where a competent valve is located.

It is the purpose of this report to present our technic and the findings in a few illustrative cases. A detailed interpretation of the venograms is being withheld until more experience has been accumulated with the method, and until interpretation can be correlated with operative findings and clinical results.

TECHNIC

The procedure, as used in a case in which definite saphenous varicosities are present, is as follows:

Under local anesthesia a longitudinal incision is made in the upper thigh over the fossa ovalis. The saphenous vein is dissected free and its tributaries are clamped cut and ligated. The vein is sectioned and the distal end ligated. A ureteral catheter is inserted for three to four centimeters into the proximal saphenous stump and the vein is ligated around the catheter. Saline is injected into the catheter to clear it of blood and the saline syringe is left attached to the catheter, which is anchored to prevent slipping. The skin incision is loosely closed and a dressing applied. Roentgenograms of the thigh are then made in the erect position after 20 cc. of 35 per cent diodrast have been injected into the catheter with minimal pressure. The injection takes from one to two minutes. Exposures are made at the completion of injection and one, two and four minutes later.

Following roentgen ray demonstration of the deep venous system the patient is returned to the operating table and the indicated surgical procedure is completed.

COMMENT

Proximal femoral venography may be expected to give information which should be a valuable supplement to the other methods of determining the cause of chronic venous insufficiency of the lower extremity. It is a simple procedure, and the results thus far obtained have been very helpful in the selection of cases for deep vein interruption. Delay in emptying and retrograde flow of the contrast medium in the upright position are findings considered to be of great significance. A variation can be made in the technic in cases having a normal saphenous system by passing the catheter into the stump of a saphenous tributary and occluding the saphenous distally with a small rubber shod clamp.

SUMMARY

A technic of proximal femoral venography in the upright position is reported. Illustrative venograms are presented.

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CAVERNOUS HEMANGIOMA OF THE LUNG*

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CAVERNOUS HEMANGIOMA of the lung is being recognized with increasing frequency. In the literature the name arteriovenous fistula of the lung is used to designate the same condition which is characterized by cyanosis, clubbing of the fingers and toes, polycythemia, a normal heart, occasional familial history of hemorrhagic telangiectasis, an obscure lung tumor or infiltration, often a murmur heard over the involved portion of the lung, and a young adult. In 1936 Bowers called attention to this condition which he noted as an autopsy finding.[1] Recently, the splendid reports of Maier,[2] Jones,[3] Moyer and Ackerman,[15] Goldman,[4] and others[4-15] have emphasized the necessity for surgical excision of the lesion. Our patient was mistakenly diagnosed as having pulmonary tuberculosis and treated for this condition several months before the true nature of the lesion in the lung was determined.

CASE REPORT

A white soldier, 20 years of age, was noted on routine roentgenogram examination of his chest, prior to discharge from the Army in October, 1947, to have an area of infiltration in the first and second right intercostal spaces anteriorly. He was asymptomatic except for a mild non-productive cough which had been present and unchanged for several years, a slight degree of dyspnea on unusual exertion, and he had noted a slight blueness of his face and hands. He was admitted to Fitzsimons General Hospital, December 4, 1947. His family history was negative and subsequent careful questioning has failed to elicit any family history of hemorrhagic telangiectasis. There was no history of contact with tuberculosis. Physical examination revealed a definite cyanosis of the face and hands and moderate clubbing of the fingers. No murmur was ever detected over the region of infiltration in the right lung. A roentgen ray film of the chest revealed an area of parenchymal infiltration in the right upper lung field in the first and second intercostal space level anteriorly (Fig. 1). This "comet" appearance is frequently seen in arteriovenous fistula of the lung.[16] He was and has remained afebrile. Electrocardiogram was within normal limits. On two occasions his sputum was reported as positive on smear examination for acid fast bacilli and the infiltration was considered as tuberculous. Treatment consisted of bed rest and 1 Gm. daily of streptomycin from January 19 to March 25, 1948, which did not alter the roentgen ray appearance of the lung lesions. Captain L. A. James, the patient's physician, suggested that this pulmonary lesion was probably not tuberculosis but rather a vascular shadow originating in the right hilar area. Studies were then directed toward proving whether the lesion was an arteriovenous fistula.

Certain studies before and after lobectomy are recorded in Tables I and II.

An angiocardiogram demonstrated an arteriovenous shunt in the right lung with slight dilatation of the pulmonary artery (Fig. 2). The Valsalva

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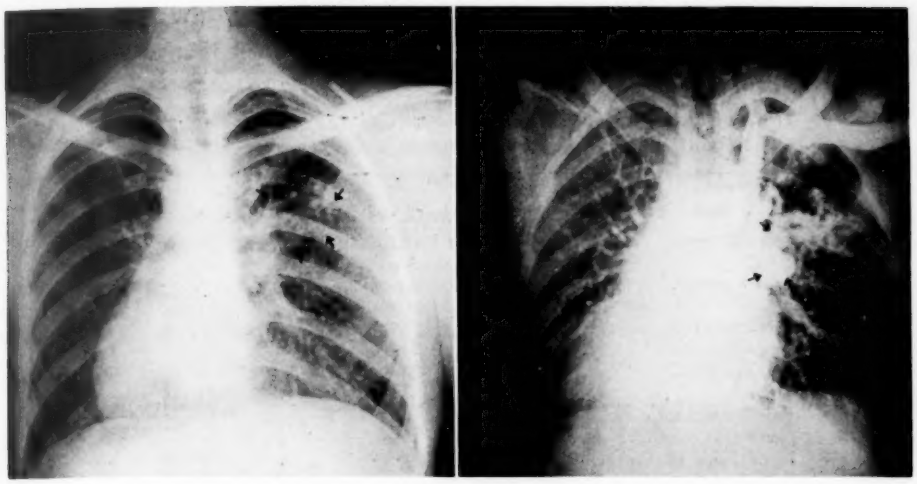

FIG. 1 FIG. 2

FIG. 1.—Infiltrativelike lesion, right upper lung field.
FIG. 2.—Angiogram, showing vascular channels in lung field.

FIG. 3.—Surgical specimen. Lipiodol of injected vascular channels.

test indicated that the lesion was of vascular origin. Fluoroscopy failed to reveal any evidence of pulsation over the infiltration. On the basis of these studies and the clinical manifestations of cyanosis and clubbing of the fingers

TABLE I.—*Observations on the Blood Volumes and Oxygen Transport in a Case of Cavernous Hemangioma of the Lung.**

	Before Operation	Three Months After Operation
Blood volumes†	7810 cc. (113.7 cc./Kg.) (normal 80-90)	5491 cc. (81.2 cc./Kg.)
Plasma volume	1851 cc. (26.8 cc./Kg.) (normal 40-45)*	2427 cc. (35.9 cc./Kg.)
Red cell volume	5959 cc.	3064 cc.
Hematocrit	76.	56
R. B. C.	8,200,000	5,600,000
Hb. (Gm.)	24	17.2
Arterial oxygen saturations		
Resting	87%	92%
Immediately after 30 eight-inch step-ups in one minute	77%‡	92%
After 5 minutes of breathing, 100% oxygen	85%	96%

* Studies carried out by Drs. Frank Princi and Joseph Holmes, of the Colorado Cardiovascular Pulmonary Laboratory, Colorado University School of Medicine, Denver, Colorado.

† Plasma volume expressed as cc./Kg. is below normal value whereas because of high hematocrit calculated blood volume is well above the normal value.

‡ "The fall in arterial oxygen saturation after exercise denotes the fact that there is a shunt somewher in the circulatory system. If there were a shunt due to inadequate transfer of gas, there would be a marke increase in saturation following the administration of oxygen. The fact that such an increase does not occu indicates that this is probably a pure arteriovenous shunt entirely within the circulatory system."—Dr. Frank Princi.

the diagnosis of tuberculosis was considered incorrect and was changed to arteriovenous fistula of the right lung. Surgical exploration of the right thoracic cavity was recommended and carried out October 21, 1948.

At operation there were a few adhesions over the posterior portion of the upper lobe and at the apex. On the posterior surface just above the interlobar fissure of the upper lobe there were many dilated venous channels. The lower lobe appeared somewhat more red than usual but no dilated venous channels

TABLE II.—*Observations on Bronchospirometry Determination in a Case of Cavernous Hemangioma of the Lung.*

	Normal		Before Operation		Three Months After Operation	
	Right	Left	Right	Left	Right	Left
Oxygen consumption	....	....	49%	51%	44%	56%
Ventilation	....	....	48%	52%	....	....
Vital capacity	....	....	50%	50%	57%	43%

were apparent on its surface. It was deemed advisable to carry out right upper lobectomy. As the lobe was elevated massive large venous dilatations in the upper lobe were seen near the interlobar fissure. These veins were the size of an average adult's index finger and were seen entering the very dilated

superior pulmonary vein. The wall of the pulmonary artery to the upper lobe was thinner than normal. No actual sinus tract connecting the pulmonary artery and the pulmonary vein forming an arteriovenous fistula was found during the course of the operation.

With the removal of the lobe immediate improvement in the cyanosis of the face was noted by the anesthetist. Plasma, rather than whole blood, was used during operation. The postoperative course was entirely uneventful. The cyanosis of the hands and fingers gradually decreased and had entirely disappeared two months after operation.

FIG. 4.—Surgical specimen. (2) Probe in postero-lateral bronchus. (3) Antero-lateral bronchus. (4) Dilated vein. (5) Area of cavernous hemangioma. (6) Artery. (7) Communication of artery and vein.

The surgical specimen (Fig. 3), demonstrated microscopically a "non-encapsulated cavernous hemangioma composed of groups of dilated, thin walled blood spaces. There was a communication between the cavernous blood spaces with a large dilated interlobular vessel that appeared to be an artery (Fig. 4). No true aneurysm is seen. The polycythemia and cyanosis were apparently due to an arteriovenous fistula; this fistula was not formed by a direct junction of

artery with vein but indirectly through the cavernous angiomatous spaces in the posteriolateral segment of the right upper lobe." There was no evidence of tuberculosis.

DISCUSSION

Venous blood in considerable quantity may be shunted from the pulmonary artery to the radicals of the pulmonary vein. This blood fails to pass through the lung and is not oxygenated and a variable degree of cyanosis and polycythemia results. These disturbed factors in the pulmonary circulation tend to increase, with accompanying increase in the cyanosis; headache may become severe, and hemoptysis may occur which may even be fatal. The absence of enlargement of the spleen aids in differentiating this condition from polycythemia vera. Malignant hemangiomas of the lung occur, but fortunately are rare and do not produce the usual symptomatology of arteriovenous fistula. The roentgenographic appearance may vary considerably. The shadow may be that of a more or less circumscribed mass or areas of increased density suggestive of parenchymal infiltration. Angiocardiograms confirm the diagnosis and it is felt that this procedure should be a prerequisite to operation. Janes[13] has reported the excision of multiple cavernous hemangiomas of the lung. Angiocardiograms would be helpful in determining the presence of such multiple lesions. The constancy of the symptomatology, laboratory and clinical findings coupled with the relative rarity of this condition makes the recording of such cases desirable.

CONCLUSIONS

A typical case of cavernous hemangioma of the lung mistakenly diagnosed and treated for pulmonary tuberculosis which was immediately cured by lobectomy is recorded.

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PRESACRAL TUMORS

CASE REPORT*

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FROM THE RIVERSIDE CLINIC

PRESACRAL TUMORS are rare and embrace a variety of types, many of which are non-malignant and amenable to surgical treatment. It is the purpose of this paper to discuss some of the clinical aspects of the more common types of these tumors and to report one case of a dermoid cyst occurring in this area.

Presacral tumors are divided by Dockerty[7] into four categories; namely, (1) congenital anomalies, (2) bone tumors, (3) neurogenic tumors, and (4) miscellaneous group. According to this author, approximately 50 such cases have been seen at the Mayo Clinic, or about 1 in 30,000 admissions. Ross[22] reports the incidence of sacral tumors as being 1 in 46,183 general hospital admissions.

CONGENITAL ANOMALIES

The origin of the congenital group of tumors[9, 16, 17, 20, 24] in this region of great developmental change is a controversial subject. It would seem that the evidence favors the origin of complex teratomas from an isolated blastomere or early budding of the blastoderm, and that the simpler teratomas and dermoids arise either from remnants of the postanal gut (Fig. 1), the proctodeal membrane, the neurenteric canal, the fovea coccygea and the coccygeal vestiges of the neural canal or from ectodermal inclusion during coalescence of cutaneous surfaces in the midline.

Chaffin[6] has suggested the explanation of congenital abnormalities in the caudal region as being due to adhesions between the rump of the embryo and the amnion. These two structures are in close proximity in the very early stages of development.

Spina bifida may be found in association with this congenital group of tumors, but usually there is no direct connection between the two. As Ewing[9] has stated, "spina bifida signifies a lack of coherence of embryonal parts and a failure of growth momentum which is favorable to spasmodic and aberrant growth of the type of budding."

Dermoid cysts are the most common congenital tumors in the presacral area and they account for a fair percentage of all presacral tumors. Whittaker and Pemberton,[24] in a study of 22 cases of tumors in this area, found that nine were dermoid cysts and one was a teratoma. Ross[22] (1948), in a review of the literature for the past ten years, found reports of 55 tumors in the sacrococcy-

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geal region, of which 15 were dermoid cysts and 11 were teratomas or teratoid tumors.

Dermoid cysts usually do not manifest themselves until adult life, the majority of patients being between 20 and 50 years of age. There are, however, a few case reports[24] where the tumor was observed at birth. Very few cases have been reported in the male.

Presacral dermoid cysts are usually silent until they reach a considerable size and cause pressure symptoms or suppurate. The common pressure symptoms are rectal or pelvic discomfort, constipation, aching in the lower abdomen, and pain in the lower part of the back. Cysts large enough to cause rectal obstruction are rare. As these tumors do not involve the sacrum, neurologic findings are unusual.

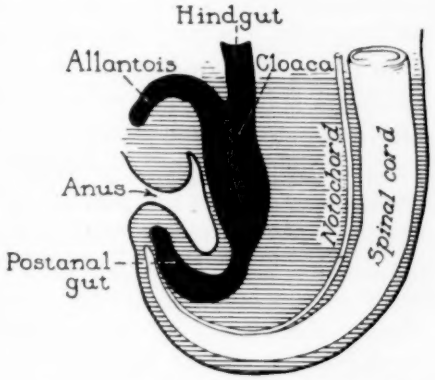

FIG. 1.—Reconstruction of the hindgut of an embryo 6.5 mm. long (modified after Keibel). Courtesy, Whittaker, L. D., and Pemberton, J. de J.: Ann. Surg., **107**: P. 98, fig. 2.

On physical examination the most common finding is a post-rectal mass felt above the anal canal ventral to the sacrum and coccyx. Usually the mass is in the midline, although it may extend laterally into the region of the buttocks. Isolated cases[20] have been reported as occurring in the perineum and ischiorectal fossa. The tumor usually does not reach huge proportions, although in one case noted[14] the mass was large enough to be palpated in the right lower quadrant of the abdomen. If infection or suppuration has not intervened, the examiner's finger in the rectum usually finds a discrete, rounded or partially lobulated, firm or cystic, non-tender tumor which is partially movable with no close attachments except in some instances to the fascia covering the sacrum. In the rare case, pressure necrosis of the adjacent rectum has occurred, with rupture into it and discharge or protrusion of hair from the anus.[15] A roentgenogram occasionally will reveal spina bifida and, in the rare instance, a calcium deposit or teeth within the cyst.

Because of traumatism, the poor blood supply to the cyst, and the low vitality of the cyst lining, these cysts frequently suppurate and many cases have been operated upon repeatedly for rectal abscess or anorectal fistula.[14, 24] If present, the sinus opening or openings are usually between the anus and coccyx, although they may be found in the ischiorectal area and, in one instance,[8] the sinus opening was found in the vagina. The nature of these complicated cases is, in some instances, not recognized, presumably because the examiner's rectal finger has neglected the hollow of the sacrum and coccyx.

In contrast to dermoid cysts teratomas usually manifest themselves at birth. Less than 100 cases of sacrococcygeal teratoma have been reported

in the literature[21] and Chaffin[6] in a study of a large group of these cases, has brought out some interesting statistics. He found that in 90 per cent of the cases a visible deformity was present at birth and that 77 per cent of the cases were female. Further, 20 per cent were stillborn, 15 per cent died soon after birth, and 65 per cent of the cases sooner or later came to operation. Of the patients who were operated upon 57.7 per cent survived. Malignant changes were observed in approximately 9 per cent of the cases.

Teratomas in this region typically have their origin posterior to the rectum and are attached to the anterior surface of the coccyx or sacrum. In the rare instance the tumor may be buried deeply and grow forward, producing a bowel or urinary tract obstruction. In the majority of cases, however, they tend to grow downward and form a visible skin-covered mass which may be quite large. On palpation it becomes obvious that the tumor consists of solid and fluid compartments. If the tumor appears to be cystic its origin from the postanal gut or neurenteric canal must be considered.[4, 20, 23]

It is important to differentiate a teratoma from the usual type of meningocele and several points have been emphasized.[6, 21] A meningocele is smaller, and does not increase in size in proportion to the infant growth, as does a teratoma; seldom displaces the rectum forward, as does a teratoma; and is covered by skin of the embryonal type. There is a communication between the meningocele and the spinal canal, and this can usually be demonstrated by expansion of the tumor when the patient coughs or cries, or by bulging of the fontanelle when the tumor is compressed. Neurologic findings in the lower extremities are more frequently associated with a meningocele. A roentgenogram will assist in making the diagnosis, particularly when bone is found in the tumor.

Of interest in this congenital group of anomalies is the rare type of anterior spina bifida associated with anterior sacral meningocele which must be differentiated from a tumor in the hollow of the sacrum. Twenty-eight such cases of anterior spina bifida associated with anterior sacral meningocele have been reported (1946).[2] The condition is not incompatible with life, as 18 of the cases were found in patients 20 years of age or older. The complaints referable to the meningocele were usually minor, constipation being the most common symptom.

BONE TUMORS

Sacrococcygeal chordomas, which are the most common sacral tumors producing pain and bone destruction, will be considered in the bone tumor group (because of their characteristic destruction of bone) although it is generally agreed that they are sui generis. The weight of evidence indicates that these tumors arise from remnants of the primitive notochord which remain in the central portion of the intervertebral discs and possibly also, as Adson *et al.*[1] have stated, from "heterotopic foci of chordal tissue anterior or posterior to the sacrum."

The chordoma is a specific tumor and "is characterized by slow growth which is inexorably progressive, a tendency to invade and destroy bone by direct extension, local recurrence after surgical excision and slight or negligible regression following irradiation. Occasionally chordoma is capable of regional and distant lymph node and visceral metastases."[11]

Gentil and Coley (1948)[11] have reported 135 cases of sacrococcygeal chordoma from a complete review of the literature, including seven of their own cases. An occasional case has been reported in infancy, but the large majority has been found between the ages of 40 and 70, and of the recorded cases Gentil and Coley[11] noted that 68 per cent were males. Trauma has been considered an etiologic factor in some cases.

Because of the place of origin of this tumor and its invasive tendencies, destruction of the sacrum slowly takes place and when the nerve roots are reached pain develops. At first it may be in the rectal and anal regions, and several cases have been reported[10, 11] where the patient had a hemorrhoidectomy performed before the proper diagnosis was reached. Intractable lower back pain, sciatic pain, urinary and rectal incontinence with objective neurologic findings are common symptoms as the bulky tumor progresses. Constipation may develop, and late in the course of the disease intestinal obstruction has occasionally been reported.

The most frequent physical finding in the presence of a sacrococcygeal chordoma is a mass, its position being determined by the direction of growth. In 18 out of 19 cases reported by Adson *et al.*[1] a presacral mass was found. These masses were densely adherent to the sacrum, irregular, and frequently nodular but not cystic. In some instances the mass extended upward to the upper level of the sacrum and displaced the rectum forward. If observed late in the course of the disease an intra-abdominal mass may be noted.

Almost every case of sacrococcygeal chordoma reveals destructive bone changes in the roentgenogram, which is a most helpful diagnostic aid, but in some instances it is necessary to conclusively establish the diagnosis by biopsy.

Ross (1948),[22] in a review of the literature on sacrococcygeal tumors in the past ten years, found 18 chordomas and four giant cell tumors reported in the bone tumor group. The ages of the patients with giant cell tumors ranged from 19 to 57 years and each presented a presacral mass. Other rare bone tumors which may present a presacral mass are Ewing's tumor, osteogenic sarcoma, myeloma, osteoma, chondroma, osteochondroma and chondromyxosarcoma. Carcinoma metastatic to the sacrum and presenting a presacral mass may also occur.[22]

NEUROGENIC TUMORS

Neurogenic tumors about the sacrum[1, 5, 12] include ependymomas, neurofibromas, neuroblastomas, and ganglioneuromas. These tumors are more uncommon than the above-described dermoid cysts, teratomas, and chordomas. All may present presacrally.

Adson *et al.*[1] (1939) reported seven ependymomas, two ganglioneuromas, and three neurofibromas from the Mayo Clinic, and, in a review of the literature for the past ten years covering tumors of the sacrococcygeal region, Ross (1948)[22] has reported six ependymomas, all of which presented presacrally.

Ependymal cells in the early embryo form a large part of the neural tube, but with development, normally remain only as the ciliated columnar cells lining the ventricle and the central canal of the cord. The sacrococcygeal ependymomas arise from these cells in the filum terminale and are localized growths which destroy bone by erosion.

Ependymomas are extremely slow-growing neoplasms which produce the same symptoms as a chordoma. However, the following differences were observed by Adson *et al.*[1] in their series of 19 chordomas and seven ependymomas: the ependymomas generally occurred in a younger age group, urinary retention with incontinence associated with pain in the buttocks and legs developed more frequently among patients with ependymoma, and the period of symptoms prior to admission of the patients with ependymoma averaged six and one-half years, whereas, with chordoma it was 13 months. Also, as a group, the masses found on rectal examination were much smaller in the cases of ependymoma and the roentgen ray findings in this group indicated destruction of bone by erosion from pressure, whereas, with the chordomas the destruction was from invasion. Furthermore, the chordomas yield very poor surgical results whereas ependymomas are frequently amenable to surgical treatment.

Tumors developing from the formative cells of the sympathetic nervous system are frequently primary in the adrenal medulla but they may arise in other areas. Neuroblastoma, a highly malignant tumor usually occurring in infancy and childhood, and the more benign mature ganglioneuroma are examples of this. These tumors are very rare in the presacral area but when they do occur may form a large mass.

A presacral neurofibroma is occasionally reported in the literature.[1, 7] This tumor may have some locally malignant characteristics.

MISCELLANEOUS GROUP

In the miscellaneous group are found those rare neoplasms which are "common to other compartments of the retroperitoneal tissue."[7] Lipoma, fibroma, fibrosarcoma, sarcoma, hemangioma, and lymphoblastoma are typical examples.

TREATMENT

The treatment of presacral tumors is surgical excision, if possible, and the approach to the posterior-inferior portion of the pelvis is the posterior route as advocated by Kraske,[13] Pearse,[19] and others. With the patient lying prone, the pelvis raised, and the thighs flexed, an incision is made from just posterior to the anus upward over the coccyx in the midline and extending

up into the sacral area as far as advisable, either in the midline or following the right or left border of this bone. The bones are exposed and the coccyx and a portion of the sacrum are removed as indicated to reach or remove the tumor. A block resection of the sacrum below the level of the second sacral segment can be carried out if necessary without serious disability to the patient.[18]

An abdominal approach to these tumors is very seldom advisable, even when the tumor can be palpated through the abdominal wall, as it is usually impossible to get at the lower portion of the tumor and hemorrhage may prove serious. Exceptions to this rule are illustrated by two recently reported cases; one by DeVoe *et al.*[7] in which a high-lying presacral neurofibroma arising from the region of the anterior border of the first sacral segment was removed through an abdominal incision. In this case the neurofibroma was holding a two and one-half month pregnant uterus in the retroflexed position. In the second case reported by Bowers,[3] a large pulsating giant cell tumor of the sacrum was successfully removed by the posterior route after ligating and transecting both hypogastric vessels and temporarily occluding both common iliac vessels through an abdominal incision.

It is agreed that teratomas in the newborn should be excised in early infancy primarily because of the deformity, the dangers of visceral obstruction, and malignant changes. Chordomas and other malignant presacral tumors should be treated by surgery followed by roentgen therapy.

Presacral ependymomas are a distinct neurosurgical problem, usually requiring an extensive laminectomy, as the tumorous tissue usually fills every crevice and foramen and frequently extends up into the lumbar canal. However, despite these locally malignant tendencies, as Adson and associates have stated, "as a whole, ependymomas are extremely slow-growing neoplasms which frequently can be completely enucleated, and an excellent prognosis can be given."

CASE REPORT

A white, 20-year-old female, was seen in consultation with Dr. Hobart M. Kelly March 19, 1948. The patient was four weeks postpartum and there were no subjective complaints.

Doctor Kelly had delivered her of two normal infants and the following are his comments regarding her obstetrical care: "The first pregnancy was terminated by spontaneous delivery January 18, 1947, of a 6-lb., 6½-oz., infant after an uneventful prenatal course and labor. The postpartum course was uneventful. She was first seen at the third month during her second pregnancy. The prenatal period was uneventful throughout. A presacral mass was first discovered during the first stage of labor February 20, 1948. To the palpating finger inserted in the rectum the tumor was noted to be in the midline posteriorly, was discrete, smooth, and rounded with a suggestion of one or two lobulations and was estimated to be approximately 3 by 4 by 5 cm. in size. Its consistency was rubbery, it was not tender, and it appeared to be partially fixed posteriorly to the lower sacrum and coccyx but anteriorly there was no evidence of intimate attachment to the posterior rectal wall. The mass did not seem large enough to produce obstruction during birth of the child and the labor proceeded uneventfully. The second stage terminated after 15 minutes with spontaneous delivery of a living infant weighing 7 lb., 10 oz., following median episiotomy. The postpartum period was completely uneventful."

The general physical examination four weeks postpartum was essentially negative except for the rectal findings. It was observed by Doctor Kelly and me that the mass, although presenting the same characteristics as described above, was now approximately 6 by 8 by 8 cm. in size (Fig. 2). Its inferior margin began about 6 cm. above the mucocutaneous junction and extended upward beyond the tip of the examining finger in the midline. Sigmoidoscopic examination to 19 cm. was essentially negative except that the posterior rectal wall was pushed forward by the extrinsic tumor.

The urinalysis and complete blood count were essentially normal. The Kahn and Kline tests were negative. Roentgenologic examination of the sacrum and coccyx revealed no abnormality of the bones.

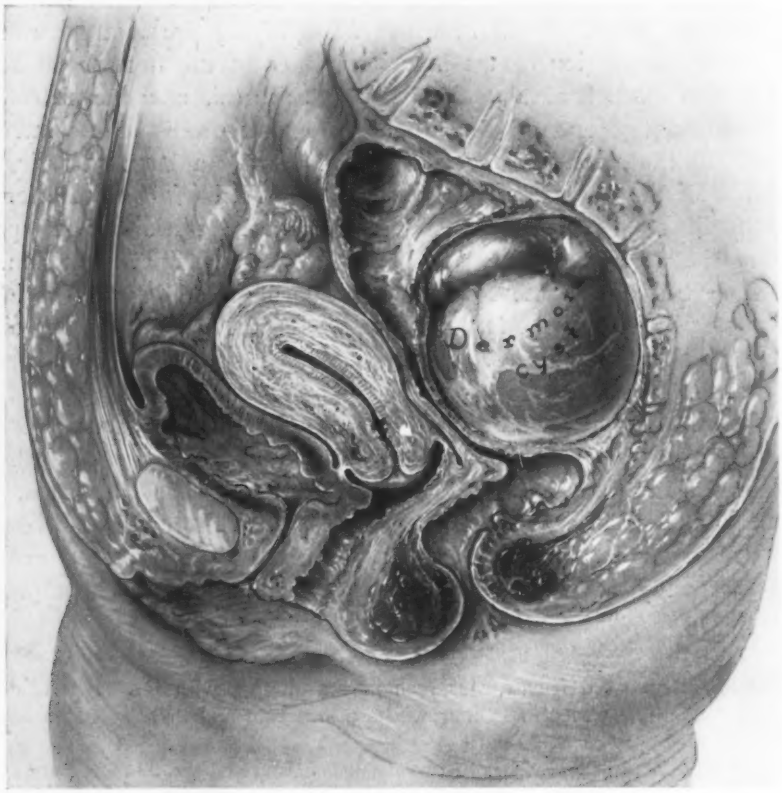

FIG. 2.—Presacral dermoid cyst.

The tentative diagnosis was a presacral tumor, probably lipoma, dermoid cyst, or neurofibroma.

On April 20, 1948, the patient was operated upon under spinal anesthesia. A longitudinal incision was made in the midline from just posterior to the rectum to the midportion of the sacrum; the coccyx was removed. Proceeding by blunt tissue dissection, a soft, encapsulated, partially lobulated, grayish cyst, about the size of a baseball, was encountered lying between the lower half of the sacrum and the rectum. The cyst was readily separated from the surrounding structures but, in order to reach its upper limits, it was necessary to sever the gluteus maximus and coccygeus muscles from the lowermost portion of the sacrum. At the upper posterior pole of the cyst a short pedicle was

found attaching the cyst to the anterior sacral fascia (Fig. 3). This was ligated and transected in the usual manner, allowing the cyst to be lifted out of the wound.

It was observed that the lower end of the sacrum presented quite a prominence which might interfere with the patient's comfort in sitting. Therefore, the distal one-inch of this bone was removed and its edges rounded. The wound was then thoroughly washed with warm, normal saline solution and closed in the usual manner, with one small Penrose drain extending up into the deeper portion of the wound between the sacrum and rectum. No attempt was made to obliterate the dead space, as the posterior rectal wall appeared quite flexible and without any limiting adhesions.

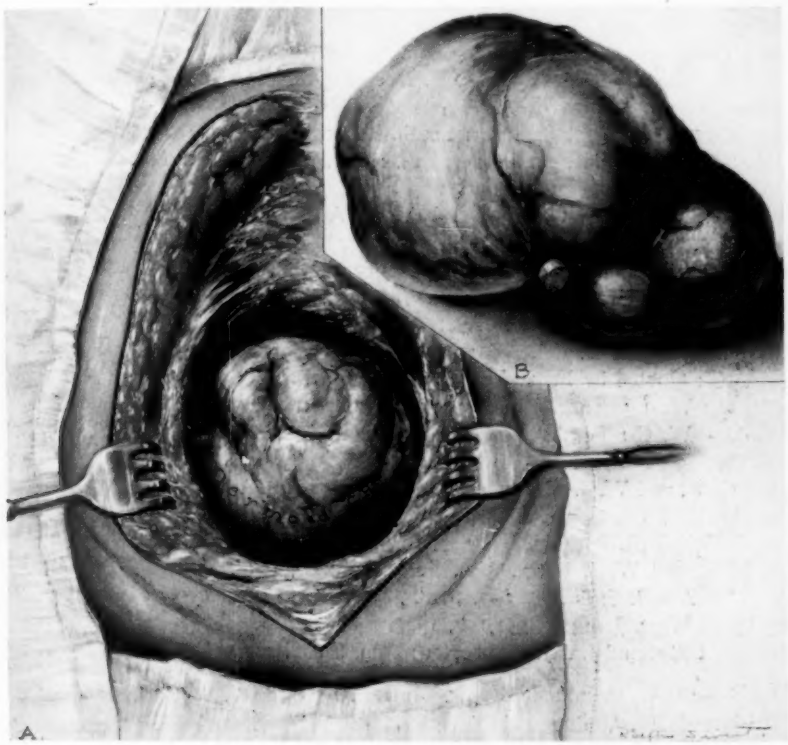

FIG. 3.—A. Posterior approach to presacral dermoid cyst. Coccyx has been removed. B. Dermoid cyst after removal.

Pathologic diagnosis of the specimen was dermoid cyst.

Early ambulation was practiced and the patient had an uneventful convalescence. She was discharged from the hospital on the sixth postoperative day and the wound was healed by the eleventh postoperative day. Rectal examination May 25, 1948, revealed a normal rectal ampulla.

SUMMARY

As a result of reports from many observers the characteristics of the relatively rare presacral tumors have been established so that a differential diagnosis can usually be made.

The origin, clinical characteristics, and treatment of these tumors have been reviewed briefly.

The case of an asymptomatic dermoid cyst which developed during the course of the second pregnancy in a young woman has been described and its surgical management discussed.

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CONGENITAL DUODENAL ATRESIA WITH MALROTATION OF THE INTESTINE

A CASE SUCCESSFULLY TREATED BY DUODENO-JEJUNOSTOMY*

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CONGENITAL OBSTRUCTION of the duodenum has been recognized since 1733 when Calder[1] first described the condition. The problem is particularly serious when encountered and terminates fatally unless surgical intervention is carried out early. The prognosis is affected materially by the rapidity with which the diagnosis is made and the proper treatment instituted. The following case of duodenal atresia is of interest because of its rarity and the associated presence of a malrotation of the intestine. The latter condition may cause a similar clinical picture, although in this instance the malrotation played no part in the obstruction.

Case Report. O. Z., a full-term Latin-American female weighing 7 5/16 pounds was delivered by Captain Charles Irwin, Station Hospital, Goodfellow Air Force Base, on October 30, 1948. She was seen in consultation by one of us (MWE) on November 2, 1948, because of repeated projectile vomiting since birth of bile-stained material following oral feedings. A meconium stool had passed on October 30 and again on October 31. Physical examination revealed upper abdominal distention. Tissue turgor was excellent due to the administration of adequate parenteral fluids since birth.

The clinical impression was atresia of the duodenum distal to the ampulla of Vater. The patient was transferred to Shannon Hospital where a roentgenogram of the abdomen revealed air in the stomach and a portion of the duodenum. There was a small amount of air in the descending colon which was considered to have been introduced by the examining finger or rectal tube.

Further roentgen ray studies were carried out after the instillation of Umbrathor†† into the stomach by catheter. Figure 1 shows dilatation of the stomach and a portion of the duodenum.

Farber's test for keratinized epithelial cells in the meconium was carried out but proved to be misleading, inasmuch as the specimen of meconium examined was improperly obtained for examination.

Operative procedure. A No. 8 soft rubber urethral catheter was inserted as an inlying gastric tube prior to the induction of anesthesia. Under drop ether anesthesia the abdomen was opened through a mid-right rectus muscle-splitting incision by one of us (GFM). On exploring the abdomen there was an excess of straw-colored fluid. The stomach and duodenum were markedly dilated and the dilatation ended sharply at the third, or transverse portion of the duodenum. There was a segment of approximately one-half inch at this area in which no evidence of intestine could be demonstrated. There was a cord-like

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†† Manufactured by Heyden Corp., New York, N. Y.

structure present and the preoperative diagnosis of atresia of the third portion of the duodenum was confirmed. The collapsed small and large intestines were in malposition. The cecum was incompletely rotated and rested in the upper portion of the abdomen in the midline. There was no evidence of bands or folds extending from this to the posterior abdominal wall which might have caused an obstruction to the duodenum. No attempt was made to restore the normal anatomic position of the cecum. A side-to-side ante-colic duodeno-jejunostomy was carried out, using two rows of sutures; an inner row of chromic 4–0 catgut sutures using the Connel-type of stitch, and a serosal row of interrupted 4–0

FIG. 1

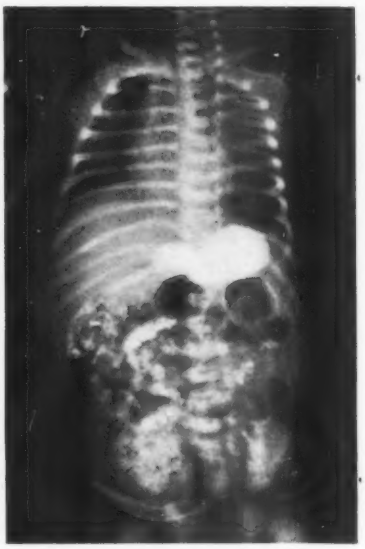

FIG. 2

FIG. 1.—Anterior posterior view of abdomen preoperatively. Air and *Umbrathor* in stomach and duodenum.

FIG. 2.—Postoperative film showing air and *Umbrathor* throughout the entire small and large intestine.

black silk sutures. When the jejunum was opened, it was inflated with normal saline solution for two reasons: first, to increase the diameter of the lumen of the collapsed jejunum to facilitate making of the anastomosis, and secondly, to rule out the presence of other obstructions lower in the intestine. The abdomen then was closed in layers with a running suture of chromic 4–0 catgut in the peritoneum and posterior rectus sheath and interrupted black silk sutures in the external rectus sheath. The skin was closed with interrupted black silk.

Postoperative course. The stomach was aspirated frequently. Fluid, consisting largely of D.A.R.,* (dextrose, amigen and Ringer's sol.) 160 cc. per Kg. of body weight, was given parenterally. On the third postoperative day dextrose water was given by mouth and the following day liquid S.M.A.† There was occasional vomiting the next 3 days, after which the child retained all feedings. The remainder of the postoperative course was uneventful. The sutures were removed on the eighth postoperative day and the child dismissed to its home on the tenth postoperative day, having regained its birth weight.

* Mead Johnson Co.

† Wyeth Co.

Postoperative roentgenograms, made on the seventh postoperative day, using Umbrathor, revealed no duodenal obstruction or delayed emptying of the stomach (Fig. 2). Weight gain and development were normal when the child was seen at the one and two months postoperative examinations.

DISCUSSION

The entire problem of congenital atresia and malrotation of the duodenum has been presented by Ladd and Gross.[2] In brief, the pathologic embryology involved occurs during the second to third month of fetal life. At this time the intestine changes permanently from a solid cord to its final form, containing a lumen lined by well defined epithelium. The failure of this to occur results in an atresia of the involved segment. The normal rotation of the intestine begins at about the tenth week of fetal life and the cecum and first portion of the colon are in the epigastrium at the eleventh week. It was probably at this stage that our case became arrested in its rotation.

The condition was suspected when the child was first seen. The diagnosis was confirmed by the use of roentgen rays, following the instillation of Umbrathor into the stomach through an inlying tube. The roentgenograms revealed a complete intestinal obstruction at the third portion of the duodenum. The radio opaque substance used for the studies was Umbrathor, an aqueous solution of thorium dioxide. The substance was mixed with equal parts of water and 10 cc. was injected through the inlying tube into the stomach. We believe this material to be without danger and far superior to barium. It may be aspirated easily and completely, if desired, at the termination of the roentgen ray studies. In addition, the possible danger of subsequent obstruction does not exist when Umbrathor is used. Farber's test[3] was carried out improperly and gave misleading information. The only meconium available for examination had to be obtained by digital means. In obtaining this specimen, stratified epithelial cells were inadvertently wiped from the cheeks of the buttocks and consequently were found on the smear. If reliance is to be placed on this test, great care must be exercised in obtaining the stool specimen. It should be obtained from the center of the stool where no contact with the skin surfaces has occurred.

Early diagnosis and operative intervention should be carried out before the infant's reserves have been depleted. Frequent aspirations of the stomach postoperatively are necessary in order to avoid undue gastric distention from the swallowed air with subsequent impingement on the intestinal anastomosis. Attention to fluid and protein balance, both pre- and postoperatively, must be carried out.

SUMMARY

1. A case is presented of congenital atresia of the third portion of the duodenum in which there was an associated malrotation of the intestine.

2. A duodeno-jejunostomy was carried out as treatment, with a successful result.

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Sterility Award

The American Society for the Study of Sterility is offering an Annual Award of $1,000, known as the Ortho Award, for an Essay on the result of some clinical or laboratory research pertinent to the field of sterility. Competition is open to those who are in clinical practice as well as to individuals whose work is restricted to research in basic fields or full-time teaching positions. The Prize Essay will appear on the Program of the forthcoming Meeting of the American Society for the Study of Sterility, which is to be held at the Sir Francis Drake Hotel in San Francisco on June 24 and 25, 1950.

Full particulars may be obtained from the Secretary, Dr. Walter W. Williams, 20 Magnolia Terrace, Springfield, Massachusetts. Essays must be in his hands by April 1, 1950.

METASTATIC CANCER OF AXILLARY LYMPH NODE WITHOUT A DEMONSTRABLE PRIMARY LESION

REPORT OF A CASE WITH A 48 MONTH LATENT PERIOD*

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IT IS CONCEDED that breast cancer can first manifest itself to the patient as a palpable mass in the axilla on the involved side. It is not exceedingly uncommon for this mass to be a lymph node, which is present without demonstrable evidence of a primary lesion within the breast, either on physical examination or on transillumination.[1] However, there were no reports uncovered in the recent literature to indicate the length of time which may elapse between the removal of the axillary lymph node involved by breast cancer and the subsequent clinical appearance of the primary lesion within the breast. For this reason the following case report is of interest, as the interval between these clinical manifestations was approximately 48 months.

CASE REPORT

A. B., a 71-year-old female, was first seen on January 22, 1949. Four years prior to this she had been operated upon and a left axillary mass excised; this mass occupied the lower portion of the left axilla. A diagnosis of metastatic cancer was made; no primary cancer was evident. She was told that no further therapy was indicated, and no additional studies were done. She was asymptomatic. One month prior to admission she went to a cancer detection clinic. Nothing was found at that time in her breasts. However, three weeks prior to entry and one week after this examination she discovered a mass in the outer quadrant of the left breast. She consulted her physician, who agreed that a mass was present and suggested that it be removed. She was referred for therapy.

The only additional significant findings were the presence of diabetes and hypertension. She had had an episode of low back pain within the last three weeks, but this had subsided spontaneously. A chest roentgenogram was normal.

She was an obese, elderly female with no pertinent findings except in the left breast. Breasts were of equal size. Nipples and areolae were normal. In the lower portion of the left axilla there was a healed incision, 4 centimeters in length. This was parallel to the circumferential border of the breast. No other abnormality was found in this area. In the axis of 2 o'clock, in the left breast, at a distance of 4 cm. from the areolar border, there was an indistinct mass measuring one cm. in diameter. This mass had ill-defined borders and was slightly tender. There was no skin attachment. The provisional diagnosis was carcinoma of the left breast.

She was advised to have an excision of the tumor in the left breast with frozen section and probable radical mastectomy. Prior to this, the histological slides of the previous axillary mass were reviewed. As these showed adenocarcinoma, it was decided to perform a radical mastectomy in any event.

Following admittance of the patient to the hospital, roentgenograms of chest, spine and pelvis were normal. On January 21, 1949, under general anesthesia, a left radical

* Submitted for publication May, 1949.

mastectomy was done; a transverse incision being used. The type of operation was that described by Haagensen.[2] There was no gross evidence of cancer in the left axillary tissue. The mass in the left breast was interpreted as adenocarcinoma on gross examination. Her postoperative course was entirely uneventful; healing being somewhat slow. This was attributed to her diabetic state. She was discharged to her home at the end of two weeks and has been relatively asymptomatic since that time.

The pathologic specimen was described as follows. "The specimen consists of a left breast measuring 40 by 29 by 5.5 cm. It is surmounted by an ellipse of skin measuring 30 by 14 cm. Near the center of the skin surface there is a grossly negative, everted nipple. In the upper outer quadrant there is an incised wound measuring 5 cm. The underlying cavity contains a blood-soaked sponge and the wall of the cavity is negative

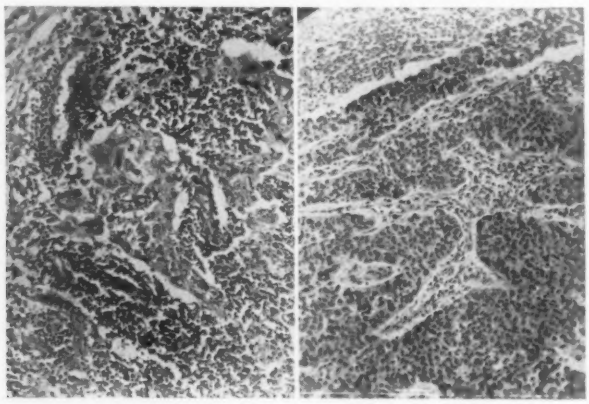

FIG. 1 FIG. 2

FIG. 1.—Carcinoma metastatic to left axillary lymph node (excised in 1945).
FIG. 2.—Adenocarcinoma of left breast (excised in 1949).

to the touch. The primary tumor which was received at the time of operation is incorporated in a mass of adipose tissue and breast tissue measuring 5.5 by 3.5 by 2.5 cm. Along one side of the specimen a tumor measuring 2 by 2 by 1.3 cm. is found. The tumor is firm, gray, and blends insensibly with the surrounding tissue. Through the firm, gray areas, foci of yellow are noted as well as several small hemorrhagic cysts. The pectoral muscles and the pectoral fascia are essentially negative. Sections through the remainder of the breast are essentially negative. Six lymph nodes are found in the lower axillary contents, and nine are found in the upper axillary contents.

Quick frozen section diagnosis: adenocarcinoma.

a. Primary tumor
b. Random sections from breast
c. Nipple
d. Upper axillary lymph nodes
e. Lower axillary lymph nodes

Microscopic Description. Sections through the primary tumor show broad sheets of anaplastic cells usually having large, irregular vesicular nuclei with granular chromatin (Fig. 2). In some cells the nuclei are condensed and hyperchromatic. Mitotic figures are scarce. The cytoplasms are pale staining, sometimes vacuolated, and the cell membranes are indistinct. Countless foci of lymphocytes are scattered through the tumor and necrotic areas are not uncommon. In some places there is an abortive attempt at duct formation. The lactiferous ducts show slight epithelial hyperplasia and in one place a small cyst lined by flat epithelial cells is noted beneath the skin of the nipple. The

papillary layer of the dermis is slightly infiltrated with lymphocytes. Random sections through the breast are essentially negative. Fourteen axillary lymph nodes were studied and none showed metastatic tumor."

The pathologist's description of the original lymph node removed four years prior and showing metastatic adenocarcinoma, is as follows (Fig. 1).

"A slide (No. 38964) representing a section of an axillary lymph node removed four years ago is almost completely replaced by tumor cells. The cells in the node are larger, more irregular, and histologically do not resemble the cells seen in the breast tumor. The different microscopic picture, the long interval, and the absence of other lymph node metastases are difficult to interpret, although the probabilities indicate that the two lesions are related.

Diagnosis. Adenocarcinoma, grade 3, of left breast, with metastasis to one (previously excised) axillary lymph node."

The microscopic slides were submitted to other pathologists, who concurred with this opinion.

DISCUSSION

There is no ready explanation for this long latent period, but it does indicate that the presence of a large metastasis and a minute primary lesion is not necessarily indicative of a rapid progression of disease. It also confirms the need for removal of the ipsilateral breast in the absence of any detectable abnormality within the breast whenever a focus of metastatic adenocarcinoma, consistent with breast origin, is found in an axillary lymph node. The minute primary may be found only by careful examination of the excised breast.

This case emphasizes the need for better methods to detect early breast cancer. This woman received a very careful physical examination one month prior to the appearance of the breast mass. Because of her history, special attention had been paid to the breast and she was examined by at least three physicians. No clinical lesion was demonstrable, although it is probable, in retrospect, that not only was the cancer present, but that it had been there and "active" for about four years. This suggests a latent period in some cases of breast cancer of sufficient length prior to appearance of clinical signs to be of practical therapeutic value if sufficiently sensitive diagnostic tests become available.

SUMMARY

A case report of cancer of the breast is presented in which a metastasis in an axillary node was excised 48 months prior to appearance of the primary lesion.

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MESENTERIC VASCULAR OCCLUSION

CASE REPORT OF SUCCESSFUL RESECTION OF ALL BUT EIGHT INCHES OF JEJUNUM AND ILEUM*

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MESENTERIC VASCULAR OCCLUSION is one of the gravest surgical emergencies. There is no typical clinical picture, so the diagnosis often is in doubt. The condition usually occurs in older patients with advanced cardiovascular disease, in whom the operative risk is great and the mortality high. The prognosis is, therefore, poor. The mortality rate in those patients subjected to resection is in the neighborhood of 90 per cent. The occurrence of mild abortive cases of mesenteric vascular occlusion is generally accepted, but this discussion is concerned only with the more familiar catastrophic type of the disease which is uniformly fatal unless treated surgically. Despite the high operative mortality, boldness on the part of the surgeon offers the patient his only hope of survival as indicated by numerous reports of successful resections of large portions of the intestinal tract.

The length of the small intestine varies greatly in man and very markedly according to its state of freshness or fixation in formalin. Morris' Anatomy[1] gives the average length of the small bowel, including duodenum, as 7 meters when freshly removed and 4 meters when fixed. However, as stated by Moynihan,[2] the length varies so that a standard measurement cannot be made. In discussing the question of removal of great lengths of small intestine he points out that the amount of bowel removed is less important than the length which remains.

Massive resections of the small intestine have been recorded since the cases of Koeberle[3] in 1880 and of Fantino[4] and Ruggi[5] in 1896, each of the latter having successfully resected over 300 cm. of small intestine. Up to the present the most extensive resections on record are those of Cogswell[6] who resected all but 14 inches of jejunum and of Grisell[7] and Meyer[8] who resected all the small bowel except 18 inches of jejunum. The case report that follows represents, I believe, the largest percentage of small bowel successfully removed, since only four inches of jejunum and four inches of ileum, in addition to the duodenum, were left.

CASE REPORT

H. K., a man of 68 years, was admitted to Mt. Sinai Hospital on April 23, 1948, with chief complaints of nausea, vomiting and severe abdominal pain. The patient was a mild diabetic, and had been under treatment for heart disease for several years. He had had a mild cerebral accident three weeks prior to admission which had left him with a slight speech defect. About 24 hours before admission he began to have profuse

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vomiting and eight hours after the onset of vomiting he complained of severe, diffuse abdominal pain and distention.

Physical examination on admission revealed an acutely ill, emaciated elderly man. The blood pressure was 180/60. The chest was fixed and emphysematous and contained many diffuse râles. The man was cyanotic and moderately orthopneic. The heart was enlarged, the rate rapid and irregular. The abdomen was distended, silent and diffusely tender. No masses were felt.

A roentgenogram of the chest was not remarkable except for cardiac enlargement. Flat plate of the abdomen showed distention of small intestine. An electrocardiogram on admission showed auricular fibrillation and evidence of severe myocardial damage. The white blood count was 25,700. Temperature on admission was 104 F. The clinical impression was intestinal obstruction with peritonitis, possibly due to mesenteric vascular occlusion. The patient was started on rapid digitalization and prepared for operation. The patient was taken to the operating room at 1 A.M. on April 24, 1948. Because of the patient's orthopnea, operation was done in a semi-sitting position. Anesthesia was uneventfully carried out on this extremely ill man by Dr. Anthony Rose, chief anesthesiologist. An endotracheal tube was passed under local anesthesia and a light ether-oxygen mixture given, supplemented by a dilute pentothal drip. Curare was given intermittently to obtain relaxation.

The abdomen was opened through a right rectus incision. A moderate amount of bloody, foul smelling fluid was found on opening the peritoneum. Almost the entire small intestine was distended, cyanotic and contained innumerable patchy areas of gangrene. No pulsations could be felt in the mesenteric vessels. No organic obstruction was present. There were no changes in the colon. Beyond the duodenum the proximal 4 inches of jejunum semed viable as did the terminal 4 inches of ileum. Resection was accordingly carried out, from a point 4 inches from the duodeno-jejunal junction to a point 4 inches from the ileo-cecal junction. The cecum and ascending colon were then mobilized to allow end-to-end anastomosis of the proximal jejunum to terminal ileum. The abdomen was closed in layers without drainage. The operation took one hour and 15 minutes and the patient withstood the procedure very well. The operative diagnosis was mesenteric vascular occlusion.

Pathologic report. Specimen consists of a portion of small bowel 258 cm. long. The bowel is thick, distended and dark purple in color.

Microscopic. Sections through various areas show varying degrees of edema, congestion and necrosis.

Diagnosis. Diffuse infarction of the small intestine.

Postoperatively the patient was placed on continuous gastric suction through an indwelling nasal tube. Fluid balance was maintained by the administration of glucose solution in physiologic salt solution and water and amigen. Vitamin B and ascorbic acid were added. Fifty mg. of heparin was given intramuscularly every 4 hours. One hundred thousand units of penicillin and 250 mg. of streptomycin were given every 3 hours. Digitalis was continued. On the second postoperative day fluids were started orally. On the third postoperative day the Levine tube was removed and he was started on a high carbohydrate, medium protein, low fat diet. His temperature gradually declined, reaching normal on the fourth day and remaining normal until discharge. Copious liquid stools commenced on the third day and were controlled with bismuth and paregoric so that by May 1, 1948, he was having only 2 to 4 semiliquid stools daily. Daily blood sugars, chlorides and carbon dioxide combining powers done during the first postoperative week were within normal limits. The patient was out of bed on May 3, 1948, in good general condition and no longer fibrillating. On May 14, 1948, barium was given by mouth and gastro-intestinal roentgenograms taken. The stomach was empty at the end of 4 hours. The anastomosis functioned normally. At 4 hours the barium had reached the mid transverse colon (Fig. 1).

The patient was kept in the hospital until May 28, 1948, for study. No evidence of tetany or of any abnormalities of his blood calcium or phosphorus were noted. Total cholesterol and cholesterol esters were normal. The prothrombin time was persistently increased and the values for thymol turbidity and cephalin flocculation were elevated. The patient was discharged on May 28, 1948, having 2 to 4 soft stools daily. He was instructed to return to the hospital in July for further study.

At home the patient again began to suffer from severe auricular fibrillation. He continued to have several semi-formed stools daily, having diarrhea only when he did not follow the prescribed high carbohydrate, medium protein, low fat diet. He entered the hospital on July 6, 1948, for follow-up study. At this time he had lost 14 pounds and was having about 4 soft stools daily. He again showed severe auricular fibrillation.

FIG. 1.—Gastro-intestinal film taken on May 14, 1948. Arrow 1 indicates duodenal-jejunal junction, arrow 2 the jejunal-ileal anastomosis and arrow 3 the ileo-cecal valve.

At this time normal values were obtained for blood sugar, urea nitrogen, chlorides and carbon dioxide combining power, calcium and phosphorus. The prothrombin time was increased and thymol turbidity was 12 units. In qualitative fecal studies the fecal nitrogen was within normal limits, fat and fatty acids were increased, and neutral fat and soaps were within normal limits. The patient had no diarrhea while in the hospital and was discharged upon completion of the studies, on July 13, 1948.

At home the patient continued to have auricular fibrillation and cardiac decompensation, which became progressively worse until his death from cardiac failure on November 25, 1948.

COMMENT

Most food absorption takes place in the small intestine. Sugars are absorbed after conversion into monosaccharides by the action of salivary, pancreatic and intestinal enzymes. Proteins, after partial cleavage in the stomach, are broken down into amino acids by the action of proteolytic pancreatic and intestinal enzymes, and are absorbed from the small intestine. Fats are broken down largely in the small intestine by the action of lipases, the most important of which is pancreatic lipase. Fat digestion and absorption is aided by the presence of bile salts. Fat is absorbed almost entirely as fatty acid and glycerol, absorption beginning in the duodenum, proceeding more rapidly in the jejunum and being complete in the terminal ileum. Severe changes in the absorption of carbohydrates, proteins and fats would be expected after removal of large portions of the small intestine. In studies of patients who have had massive small bowel resections it has been found that carbohydrates are well absorbed, about 75 per cent of protein is absorbed and about 55 per cent of fat. In a case reported by West and his associates[9] fecal fat consisted in large proportion of free fatty acids, indicating good fat digestion but poor absorption. Tetany and low blood calcium values have been reported following massive intestinal resections, presumably due to the large amounts of free fatty acid in the stool carrying away calcium as calcium soap. In the case reported here the fecal findings corresponded closely to those found by West. However, our patient showed no evidence of tetany or did he have any change in blood calcium figures. No supplementary dietary calcium was needed. All blood chemical determinations were within normal limits with the exception of certain liver function studies. The increased prothrombin time, thymol turbidity and cephalin flocculation in this patient were probably due to hepatic changes resulting from chronic passive congestion secondary to long-standing cardiac decompensation. The diarrhea was well controlled by a high carbohydrate, medium protein, low fat diet.

SUMMARY AND CONCLUSIONS

A case has been presented of a 68-year-old man who developed gangrene of the entire small bowel with the exception of the duodenum and four inches each of ileum and jejunum. Resection and primary anastomosis was carried out successfully. The patient survived seven months, ultimately dying of cardiac decompensation. Studies were carried out which indicated that the major postoperative digestive difficulties concerned absorption of fat. Protein and carbohydrate metabolism did not show any remarkable changes.

Mesenteric vascular occlusion is a catastrophic abdominal emergency. Under conservative treatment the mortality in patients with more than a minor abortive area of occlusion, is in the neighborhood of 100 per cent. While the operative mortality is high, wide, radical resection and restoration of intestinal continuity offers the only hope of survival. The case presented in this paper and others quoted attest to the occasional success of radical surgical treatment.

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Editorial . . .

Psychiatry and Surgery

The final goal of Surgery and that of Psychiatry are identical. The drives, motivations and capacities present in any one individual which contribute to his becoming a surgeon may vary over a wide field. The patient who seeks the help of the surgeon has, however, one fundamental motivation. He is in some way convinced that the result of the surgical procedure to be performed will improve his capacity to deal successfully with his own life situation. In the sense of its service to humanity, the final objective of surgery is, then, the improvement of the patient's capacity to adjust to his surroundings. The nature of the profession of surgery makes it possible for some who practice it to attain the humanitarian objective without even being aware of it or consciously striving toward it. Having recognized the final objective of their labors, others have expressed the opinion that their efficiency would be enhanced by a clearer concept of the patient as a living, experiencing person, and of the role the patient's experiences play in the progress of illness. They express the opinion that an improved psychiatric orientation would serve to make surgery more effective as an asset to humanity.

Psychiatric orientation is an important part of the development of the individual surgeon's philosophy concerning the meaning of his profession, and the way it fits into the culture, and contributes to the common good. If he sees the patient as an experiencing human being and exerts a continuous effort to understand the patient's surgical problem in terms of human experience, he may be aided in the fulfillment of his service to humanity by acquiring an improved understanding of human behavior as interpreted by psychiatry. If, on the other hand, he sees the patient as a kind of detached bearer of a machine, brought in to have its parts mended, his philosophy will preclude his recognition that psychiatry has anything to do with the task he performs. The surgeon who subscribes to the former philosophic approach will likely experience considerable frustration if he turns to ordinary psychiatric literature for assistance in understanding his patient as a personality. The literature is voluminous and controversial, and too frequently employs a jargon that at times defies translation, even by those who use it. He may also be confounded by his own habitual tendency to think of the personality as a thing apart and separate from the living anatomy with which he is so tangibly familiar. The dichotomy of mind and body is so much a part of the basic philosophy on which our culture is built that the most cautious may revert to it even while assuring themselves that they have discarded it. Explorations of the psychiatric literature and fruitless appeals to his psychiatric friends for help may leave the surgeon with a feeling of futility. He may consider that appropriate sources have failed to supply a formula or way of

thinking that would assist him in understanding his patient more completely. Frustrated by failure to gain the assistance he needs from the psychiatrist, he may join his colleague, who subscribes to the alternate philosophic approach. Identifying himself with those who see themselves as technicians or menders of parts, he may take refuge in the dichotomy and defend his position by declaring that his professional duty ceases with the skillful performance of his technics, and that all other matters are entirely the responsibility of the patient. Some seem able to occupy this position with considerable comfort. Others who try are disturbed by the frequency with which the way the patient experiences his surroundings, has direct effect upon the results of their applied technics.

It is no more reasonable to expect the surgeon to be completely trained in psychiatry than it is to expect the doctor who is psychiatrically trained to be prepared to function as a surgeon. Familiarity with some fundamental psychiatric principles will, however, aid the surgeon in the performance of his own function in several ways. Some of these are:

1. *The general approach to the patient.* A kind of thinking about the patient which first considers the whole living, experiencing entity, and then considers the complaint and the pathology productive of it, in terms of their meaning to that living, experiencing entity, will not only contribute to attaining the final objective of surgery, but will lend a new fascination to the surgeon's own experience.

2. *The use of the psychiatric consultation.* Some competence in the understanding of the human personality and of human experience is essential in order to utilize the assistance the psychiatric colleague has to offer. The simple matter of gaining the patient's cooperation in seeking the collaboration of a psychiatrist is sometimes so baffling that the surgeon avoids it against his better judgment. More baffling may be the problem of interpreting the report the psychiatrist returns after having examined the patient. While the psychiatrist making the report should take into consideration the surgeon's point of view, the surgeon will profit more from the psychiatric consultation if he devotes some effort to understanding how the psychiatrist thinks and why he thinks that way.

3. *The recognition of the emotional and experiential factors which contribute to the etiology of conditions which come under the observation of the surgeon.*

4. *The recognition of emotional and experiential factors which directly effect the result of treatment, favorably or unfavorably.*

5. *The evaluation of the patient for whom elective surgery is considered.*

6. *The planning and preparation of the patient's rehabilitation program.*

7. *The surgeon's evaluation of himself in the treatment situation related to any particular patient.*

Psychiatry in surgery will have its greatest value when used by the surgeon himself as a kind of orientation in understanding the patient as a whole

human being. The surgeon will find the application of a psychiatric orientation facilitated if he will:

1. *Recognize the personality of the patient as the total integrated physiologic activity of a biologic entity.* In this way the behavior of the individual can be seen as the product of the function of structures just as "real," "physical," or "organic" as are those that produce the measurable and palpable signs of morbid anatomy. In this connection, it is important to avoid the use of the terms "functional" and "organic" as antonyms. These terms tend to perpetuate the philosophy of the dichotomy, and on close examination lose distinctive significance. Function is the activity of organs and is therefore "organic." Any organ that is alive functions, and is therefore "functional."

2. *See each patient as a separate biologic entity, experiencing its particular physiologic activity in the medium of its own specific surroundings.*

Having accomplished this the surgeon will deal with each patient as a person he knows in a situation he understands, and will then be in a better position to achieve the final goal of surgery.

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BOOK REVIEWS

BRITISH SURGICAL PRACTICE. Edited by Sir Ernest Rock Carling and J. Paterson Ross, Butterworth & Co., London, and C. V. Mosby Co., St. Louis, Mo., Vol. IV.

This volume is the fourth of a series edited by two British surgeons. Subjects are taken up alphabetically; those from facial palsy to hiccup are included herein. Each subject is written by a different author or authors, who have been chosen primarily because of their interest and wide experience in the subject assigned to them.

Each section is preceded by a table of contents enabling the reader to determine at a glance what main headings are included in the section. In fine type along the margin are listed important points included in the text. The type is well chosen to differentiate the various headings and subheadings. The paper is a good quality, thus reproducing the illustrations well.

The authors have presented their material in good pedagogical form, each devoting space to etiology, surgical anatomy, pathology, diagnosis, treatment, etc. No attempt is made to give historical sketches of old, obsolete operations. In general, the author describes only the operative procedure found to be most successful in their hands, thus conserving the reader's time. Of the major subjects, fascial grafts, fistulae in ano, focal epilepsy, gallbladder, gangrene, gastro-colic fistula, heart and pericardium, and hernia are particularly well covered. Operations on the great vessels are not included in this volume.

This volume contains a lot of valuable information which will be found very useful to the general practitioner and surgeon. Even though there are several volumes in this series, surgical subjects are so numerous that the editors and authors have found it necessary to condense the material quite sharply. For example, only 21 pages are allowed for the gallbladder and biliary passages, 15 pages for the hand, and 67 pages for fractures, dislocations and allied injuries. The illustrations are well chosen and are clearly reproduced, but a greater number, particularly in the field of operative technic, would be welcome.

WARREN H. COLE, M.D.

THE 1948 YEAR BOOK OF GENERAL SURGERY. Edited by Evarts A. Graham, A.B., M.D., pp. 695, ills. 190, 1949, The Year Book Publishers, Inc., Chicago.

As usual, The 1948 Year Book of General Surgery presents an excellent and brief review of each of more than 400 of the best articles in surgical literature published in 1948. The editor makes numerous comments and suggestions which are very interesting and educational.

There were few new operations described in the year's literature, although articles suggesting refinements of previously devised operations were relatively frequent. The greatest surgical progress during the past year has been in the better understanding of the pathologic physiology and biochemistry involved in surgical diseases, and applying this knowledge not only in the operating room but, even more important, during the pre- and postoperative periods. This has resulted in decreasing the surgical mortality and postoperative morbidities. The most striking effect of this is noted in such transthoracic operations as resection of the esophagus, and surgery of the heart and major vessels.

For ease of reading and reference, the articles reviewed in this Year Book have been divided into 28 chapters, classified by subject. The chapters on Metabolism-Electrolytes and Thorax are outstanding. This yearly publication should be read by every surgeon.

L. W. PETERSON, M.D.